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Diallel analysis of the latent period of stripe rust in wheat

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Abstract A half diallel was made amongst five wheat (*Triticum aestivum* L.) genotypes of which one was susceptible, while the others had adult-plant resistance, to stripe rust (*Puccinia striiformis* West.). The five parent and ten F_1 progeny were grown in the glasshouse and were inoculated with three rust pathotypes at the seedling stage. The latent period was measured on the first leaf. Two procedures were used to analyze the half diallel. Both methods showed that the average effects of alleles were of much greater importance than was dominance in conditioning resistance in response to two of the pathotypes, while for the third pathotype dominance was important. Resistance was conditioned by partial dominance for two pathotypes whereas for the third it was determined by full dominance. Broad-sense heritabilities range from 60–73% and the number of genes involved was different (from 1 to 4), depending on the pathotype.

Key words Wheat · *Puccinia striiformis* · Stripe (yellow) rust · Diallel analysis · Latent period

Introduction

Stripe rust (yellow rust) caused by *Puccinia striiformis* West. f.sp. *tritici* was first reported in Australia in October 1979 (O'Brien et al. 1980) and first appeared in New Zealand in 1980 (Beresford 1982; McIntosh and Wellings 1986). In New Zealand, crop losses in susceptible cultivars were as high as 60% (Beresford 1982).

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Exploitation of stripe rust resistance genes may be economically feasible, and environmentally safe in contrast with chemical control. Cultivars that contain only specific major genes for resistance, whether for seedling resistance or adult-plant resistance, are vulnerable. Breeders have turned their attention to other forms of resistance, such as adult-plant resistance (which may be durable) or slow rusting, which reduces the rate of epidemic development by reducing pustule density and pustule size, with a longer latent period (Parlevliet 1979, 1985). In other rusts it has been reported that the latent period is the easiest component to analyse (Shaner 1980; Shaner and Finney 1980) since it can be measured with least error (Kuhn et al. 1978; Shaner et al. 1978; Shaner and Finney 1980). However, there is no published information on the inheritance of the latent period in response to stripe rust, although variation for the latent period has been reported for stripe rust by Duhne (1977, quoted by Parlevliet 1985), Park and Rees (1989), and Cromey (1992). The object of the present study was to investigate the genetic control of the latent period of stripe rust resistance.

Materials and methods

Four wheat genotypes which exhibited resistance to stripe rust and one susceptible cultivar (Briscard, Domino, Otane, Ruapuna and Tiritea, respectively) were intercrossed in a half-diallel mating system. Three cultures of *P. striiformis* (106E139A-, 111E143A- and 232E137A-) from the rust culture collection of the Crop and Food Research Institute at Lincoln, New Zealand, were used in this investigation. Pathotype nomenclature follows the system described by Johnson et al. (1972), using the suffix introduced by Wellings and McIntosh (1990). Uredospores of the three cultures were increased at three different times on the susceptible wheat cv Tiritea grown in isolation in the glasshouse. Each day, inoculum was collected, partially dried, and then sealed in plastic-lined aluminium foil bags and stored in an ultra-low freezer at − 70 °C. Inoculum of each pathotype was heat-shocked by immersing in warm water (42 °C for 4 min) before use.

The parents and F_1 progenies were planted in 10-cm pots containing potting mixture (sand, peat and osmocote 5:2 parts and 250 g, respectively), using three diallel experiments, one per pathotype, in

three different places to prevent any contamination. The pots were placed in the glasshouse with a 15-h daily photoperiod at 15 ± 2 °C. Inoculation was carried out when the first leaf was fully expanded and the second leaf was about half the length of the first. For inoculation, all pots were sprayed as uniformly as possible using an atomizer containing a spore suspension in distilled water with one drop of Tween 20 per litre and were then left in a darkened moist chamber for 24 h at 10 ± 1° C. Daily assessments from the 7th day after inoculation were made for the latent period (days from inoculation to first pustule eruption) by checking all leaves for visible pustules. Leaves with pustules were tagged, the data recorded, and these leaves omitted from further checking. Daily assessment continued until pustules were present on all leaves.

The parental and F_1 data were analyzed using both the graphical technique of Mather and Jinks (1982) and the combining ability method 2, model I (fixed effects) of Griffing (1956). In the first case, the variance of each array (V_r) and the covariance of each array with the non-recurrent parents (W_r) were calculated for the linear regression analysis of W_r and V_r. The genetic components of variation were estimated and some of the genetic statistics required for the diallel cross data (Mather and Jinks 1982) were also estimated. In the second case, the data were analyzed to estimate general and specific combining ability effects (g.c.a. and s.c.a., respectively, Griffing 1956). The model is as follow:

$$x_{ij} = u + g_i + g_j + s_{ij} + (1/bc)\Sigma_{k=1}\Sigma_{1=1}e_{ijkl}$$

where u is the population mean, g_i and g_j are the general combining ability effects for the i_{th} parent and j_{th} parent, respectively, s_{ij} is the specific combining ability effect of the cross between the i_{th} and j_{th} parents, e_{ijkl} is the effect associated with $ijkl_{th}$ individual observation, b is the number of blocks, and c is the number of plants per plot.

Results

The differences among genotypes (Table 1) suggest that much genetic variability was present for the latent period in the genotypes studied, and diallel analysis was warranted for it.

All the diallel statistics for the latent period for the three pathotypes are presented in Table 2. The $W_r + V_r$ and $W_r - V_r$ values were not significant. The regression coefficients ($\beta - 1$) were significant for pathotype 111E143A-, whereas it was not significant for the other two pathotypes. Additive genetic variance (D) was greater than the dominance genetic variance (H_1 and H_2) for pathotypes 106E139A- and 111E143A-, whereas for pathotype 232E137A- dominance variance (H_1) was greater. Estimates of F were positive for all three pathotypes, indicating the inequality of gene frequencies with an excess of dominant over recessive alleles. The degree of dominance, $\sqrt{(H_1/D)}$, indicated that partial dominance for pathotype 111E143A- was present while for pathotype 232E137A- full dominance was observed. It was not calculated in the case of pathotype 106E139A- due to its negative value. The gene frequencies (0.21 and 0.15) indicated the inequality of genes for increasing and decreasing the latent period. The proportions of positive to negative alleles were 1.11 and 0.59 for pathotypes 111E143A- and 232E137A-. In the case of the first of these pathotype, the $0.5\,F/\sqrt{[D(H_1 - H_2)]}$ ratio was found be approximately equal to 1, suggesting that the observed dominance was consistent for all the loci, rather than with variable degrees of dominance at different loci. The proportion of dominant to recessive alleles over all parents was respectively 2.63 and 2.17 for pathotypes 111E143A- and 232E137A-. Estimates of $0.5\,F/\sqrt{[D(H_1 - H_2)]}$ and the proportion of dominant to recessive genes were trivial for pathotype 106E139A-, due to the unimportance of dominance. Narrow-sense heritability values were less than broad sense. The ranges of broad-sense and narrow-sense heritability for the latent period in response to the three pathotypes were 60–73% and 30–52%, respectively. The correlations between common parent means and $W_r + V_r$ for each array were significant and indicated that the distribution of dominant to recessive alleles was correlated with the common parent phenotype for all three pathotypes, i.e., positive and significant coefficients indicated that parents having a lower phenotypic mean (short latent-

Table 1 Duncan's multiple ranges of five cultivars and their progenies for the latent period in response to three pathotypes of stripe rust

Cultivar	Pathotype		
	106E139A-	111E143A-	232E137A-
Tiritea	11.7 a	11.7 a	11.0 a
Domino/Tiritea	12.1 ab	12.0 a	11.6 bcd
Otane/Tiritea	12.5 bc	11.9 a	11.2 ab
Otane	12.5 bc	12.2 a	11.6 abcd
Briscard/Tiritea	13.2 cd	11.8 a	11.3 abc
Ruapuna/Tiritea	13.0 cd	12.4 ab	11.1 ab
Domino/Otane	12.8 bcd	12.3 ab	11.6 bcde
Ruapuna/Otane	13.3 de	12.3 ab	11.3 abc
Briscard/Otane	13.1 cd	12.4 ab	12.2 efg
Ruapuna/Domino	13.1 cd	12.3 ab	11.3 abc
Domino	13.5 de	13.0 bc	11.6 abcd
Ruapuna	14.8 gh	13.5 c	11.4 g
Briscard/Ruapuna	14.3 fg	12.3 ab	11.8 cdef
Briscard	15.1 h	13.3 c	12.1 defg
Briscard/Domino	14.0 ef	12.2 a	12.2 fg

Table 2 Genetic statistic for the latent period of stripe rust in five cultivars of wheat

Statistics	Pathotype[a]		
	106E139A-	111E143A-	232E137A-
$W_r + V_r$	ns	ns	ns
$W_r - V_r$	ns	ns	ns
$\beta - 1$	0.75 ns	3.71**	1.42 ns
D	2.02	0.47	0.27
H_1	−0.31	0.38	0.31
H_2	0.38	0.31	0.19
F	0.67	0.38	0.21
$(H_1/D)^{1/2}$	–	0.89	1.08
$u\bar{v}$	–	0.21	0.15
$0.5F/[D(H_1 - H_2)]^{1/2}$	–	1.11	0.59
Dom./rec. genes	–	2.63	2.17
h^2_{BS}	0.67	0.60	0.73
h^2_{NS}	0.52	0.30	0.48
$r(P_r, W_r + V_r)$	0.82	0.77	0.88
K (effective factors)	1.6	3.9	0.6

[a] ns, not significant; **, $P < 0.01$

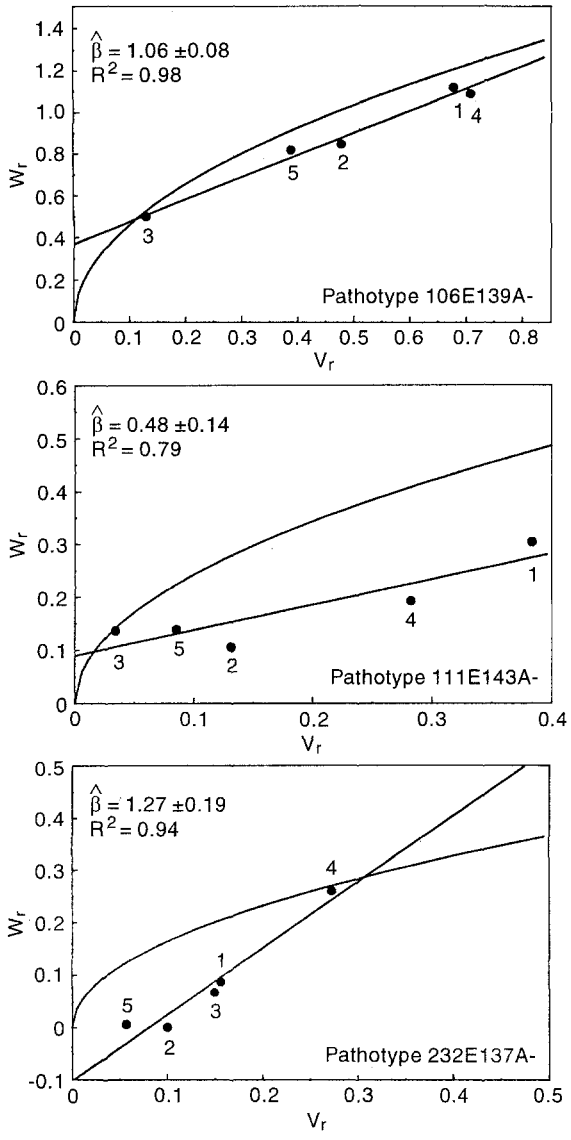

Fig. 1 The W_r/V_r graphs of the latent period from crosses of five cultivars of wheat (*1*, Briscard; *2*, Domino; *3*, Otane; *4*, Ruapuna and *5*, Tiritea) in response to three pathotypes of stripe rust

period) were dominant to those having a higher phenotypic mean. The effective factors for pathotypes 106E139A- and 111E143A- were 1.56 and 3.90, but was less than 1.0 for pathotype 232E137A-.

The graphic analysis (Fig. 1) for pathotype 106E 139A- shows that Briscard and Ruapuna contained most recessive alleles, while Otane contained most dominant alleles. Tiritea and Domino were intermediate. For pathotype 111E143A-, the order of parents was the same as for the first pathotype, but Ruapuna, Domino and Tiritea contained fewer recessive alleles. For pathotype 232E137A-, Ruapuna contained the highest proportion of recessive alleles whereas Tiritea had the highest proportion of dominant alleles. The rest of the parents were intermediate between them. The coefficients of determination were 98.2, 79.2 and 94.0% for pathotypes 106E139A-, 111E143A- and 232E137A-, respectively.

Table 3 Mean squares of general and specific combining ability for the latent period of three pathotypes of stripe rust

S.O.V.	*df*	Pathotype		
		106E139A-	111E143A-	232E137A-
GCA[a]	4	2.9**	0.5**	0.4**
SCA[b]	10	0.2**	0.2**	0.1**
Error	233	0.03	0.02	0.03
Ratio[c]		0.97	0.83	0.89
h^2_{BS}[d]		0.99	0.98	0.97
h^2_{NS}[e]		0.96	0.82	0.86

[a] General combining ability
[b] Specific combining ability
[c] $2MS_{GCA}/(2MS_{GCA} + MS_{SCA})$ (Baker 1978)
[d,e] 4,5 Broad- and narrow-sense heritability, respectively

The intercept of the regression line for pathotypes 106E 139A- and 111E143A- was above the origin, and for pathotype 232E137A- was below the origin, indicating partial dominance and overdominance, respectively.

In the analysis of the latent period of each pathotype, the mean squares for GCA are larger than those for SCA (Table 3). Both GCA and SCA are highly significant. The ratio, $2MS_{GCA}/(2MS_{GCA} + MS_{SCA})$, was 0.97, 0.83 and 0.89 for pathotypes 106E139A, 111E143A- and 232E137A- respectively, indicating the relative importance of additive to nonadditive effects. Narrow-sense and broad-sense heritabilities ranged from 0.82 to 0.99 for all pathotypes. For the three pathotypes, the GCA for Briscard, Ruapuna and Domino were positive, suggesting they are suitable parents for obtaining a longer latent period; whereas for Otane and Tiritea they were negative (Table 4). The ranking of cultivars according to their GCA and performance (Table 1) was the same. The SCA effects for the latent period of pathotypes 106E139A-, 111E143A- and 232E137A- showed respectively that 1, 0 and 2 combinations had significant positive value (Table 4). The highest SCA was shown by the combinations Otane × Tiritea for pathotype 106 E139A-, Ruapuna × Tiritea for pathotype 111E143A-, and Briscard × Otane for pathotype 232E137A-, suggesting the presence of dominance for the longer latent period in those hybrids.

Discussion

Knowledge of the mode of inheritance or of the genetic architecture permits an overall assessment of the probable effects of selection on any generation. Amongst all mating designs, diallel matings, especially half diallel (Kearsey 1965), provide a simple and convenient method for estimating genetic parameters. Diallel analysis is a powerful technique for obtaining a rapid, overall picture of the genetical structure of a large number of parental lines, i.e., overall degree of dominance of the relative dominance properties of the parents (Jinks 1954).

Table 4 Estimates of general (on diagonal) and specific (above diagonal) combining ability for the latent period in response to three pathotypes of stripe rust

106E139A-	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	0.74**	0.05ns	−0.52**	−0.24*	−0.02ns
Domino		−0.10**	0.07ns	−0.63**	−0.29**
Otane			−0.42**	−0.05ns	0.41**
Ruapuna				0.54**	−0.08ns
Tiritea	$SE_{GCA} = 0.0350$	$SE_{SCA} = 0.0930$			−0.77**
111E143A-	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	0.14**	−0.42**	0.05 ns	−0.55**	−0.35**
Domino		0.09*	−0.05ns	−0.42**	−0.05 ns
Otane			−0.14**	−0.25*	0.07 ns
Ruapuna				0.31**	−0.15ns
Tiritea	$SE_{GCA} = 0.0320$	$SE_{SCA} = 0.0849$			−0.39**
232E137A-	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	0.28**	0.30**	−0.33**	−0.20**	−0.28**
Domino		−0.04ns	0.04ns	−0.44**	0.32**
Otane			−0.04ns	−0.38**	−0.03ns
Ruapuna				0.07**	−0.23**
Tiritea	$SE_{GCA} = 0.0220$	$SE_{SCA} = 0.0572$			−0.35**

Biometrical analyses of the diallel for the latent period, in response to three pathotypes, showed that additivity was of major importance in conditioning this trait, because the D components were relatively large for pathotypes 106E139A- and 111E143A-, while for pathotype 232E137A- dominance was important. It should be noted, however, that the preponderance of additivity found in these experiments did not necessarily indicate that dominance was lacking. Additivity is the variance of average allele effects and therefore does not indicate an additive action of genes. That is, additive genetic variance in no way implies that dominance and/or epistasis are absent (Falconer 1981). It has been commented that dominant genes, with the exception of those that exhibit overdominance, have at least half of their effect estimated as additive (Mather and Jinks 1982).

Because there are, as yet, no known Yr gene(s) in the cultivars employed, it is important that in certain cultivars the mode of gene action is changed by using different pathotypes. This means that there is an interaction between genes in the cultivars with those in the pathotype. If a single gene controlled resistance, there would be considerable interaction between the genotype and the pathotype, but if resistance was controlled by more than one gene (polygenes), the interaction would not be great. Differential interaction of partial resistance was reported with Parlevliet (1977), but he mentioned that this type of resistance is under polygenic control and was quite stable.

Negative estimates of H were calculated in response to pathotype 106E139A-, indicating that dominance was trivial. Variance components are conceptually positive, but negative estimates of variance components are common in research because of sampling distributions. Possible causes of negative estimates have been discussed by Searle (1971) and Reeder et al. (1987). Where positive values of H_1 occurred, estimates of the degree of dominance $[(H_1/D)^{1/2}]$ were calculated. These degrees of dominance can be reliable when there is no epistasis. However, it is important to realise that, in the presence of epistasis, the degree of dominance will be biased upwards (Hayman 1954; Jinks 1954). Correlated gene distribution can also bias the dominance upwards and so could be responsible for inflating the apparent degree of dominance.

The narrow-sense heritabilities were high, or moderately high, for the latent period, indicating that selection for resistant genotypes will be effective. This agreed with the importance of additivity. The number of genes involved in resistance was 1–2, 3–4 and 1 in response to pathotypes 106E139A-, 111E143A- and 232E137A-, respectively. Probably, these differences are due to differences in virulence genes between these pathotypes. It should be noted that, in a diallel cross, the number of effective factors is not as clear. Firstly, our estimate must of necessity come from the H statistics which are undoubtedly inferior to those obtained from D statistics. Secondly, any deviation from a random association of gene differences throughout the parental lines leads to a further minimizing of the estimate (Jinks 1954). Unfortunately the number of effective factors rarely equals the gene number (Mather and Jinks 1982) and so must be used with caution.

The dominant alleles (short latent period) were more frequent than the recessive alleles in these experiments because, in the presence of unequal gene frequencies, the sign and magnitude of the F value can be used to

determine the relative frequencies of dominant to recessive alleles in the parental population, and the variation in the dominance level over loci. The value of F was positive whenever dominant alleles are more frequent than recessive alleles, irrespective of whether or not the dominant alleles increases or decreases.

In response to pathotype 111E143A-, there was a lack of agreement between the analysis of variance ($W_r + V_r$) and the result of the significance test for the departure of the regression slope from unity. It can be concluded that epistasis and/or correlated gene distribution was present, although they were relatively unimportant. Mather and Jinks (1982) noted that the lack of agreement between these results indicates that the suitability of the model is equivocal. Furthermore, the evidence for disturbance is generally weak. Therefore, it is appropriate to proceed with the analysis and estimate genetic components and other statistics with presumably some bias. The $W_r + V_r$, P correlation coefficients were generally high and significant for most characters. It can be concluded that there was directional dominance in conditioning the short latent period.

If there is no epistasis or correlated gene distributions, then graphical analysis of W_r/V_r can be more reliable. But in the presence of non-allelic interactions, the graph is sensitive to interactions. Also it has been demonstrated that gene dispersion and association cause the W_r/V_r graph to deviate from a straight line of unit slope in characteristic ways which have a superficial similarity to the effects of complementary and duplicate interactions, respectively (Mather 1967). In the present experiments genetic diversity among parents was demonstrated by the scatter of the parental array points along the regression line of the W_r/V_r analysis. Reversal of the mode of gene action was observed; for example, Otane showed dominance against all pathotypes while the rest of cultivars had a reversal of dominance (Fig. 1). The reversal mode of gene action has been reported by other workers (see Lupton and Macer 1962). One possible conclusion is that the reversal of dominance could be due to a higher level of inoculation pressure between experiments or to environmental differences. While these experiments were carried out at different times, an attempt was made to have otherwise identical conditions.

The general combining ability, describing the average performance of a line in hybrid combinations, and which contains mainly additive effects, was found to be the major component of variation. Also, significant specific combining ability, which is a measure of the deviation of crosses from the value expected on the basis of the performance of the parents, and is composed of dominance plus interallelic interaction or epistasis variance, was present in all cases. Also, in all cases the ratio proposed by Baker (1978) was close to unity, suggesting that additive effects were more important than nonadditive effects for resistance to stripe rust. Judging by the ratios expressing the relative importance of general combining ability and specific combining ability, additive variance was of major importance. This signifies that progeny performance can be effectively estimated on the basis of general combining-ability effects, as the reaction to the disease appears to be uniformly transmitted to all offspring. It should be possible, therefore, to manipulate these resistance genes in a breeding programme because of the high level of additive gene effects and high heritability. This experiment is in agreement with other reports (Kim and Brewbaker 1977; Krupinsky and Sharp 1978).

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