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Inheritance of Latent Period of Stripe Rust in Wheat

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ABSTRACT

Genetics of slow-rusting resistance to yellow rust (*Puccinia striiformis* West.) was studied by a half-diallel design using five wheat genotypes, Bolani (susceptible), Brock, Domino, Elit-Lep, and Kotare.The parents and ten F_1 progenies were evaluated in the greenhouse by four pathotypes 134E134A+, 140E72A+, 174E174A+ and 230E15A+.The latent period was measured as the number of days from inoculation to the appearance of the first pustule. For each pathotype a randomized complete block design was used and data were analyzed by methods of Griffing and Hayman. Positive and negative degrees of dominance were observed for each pathotype that showed the reversal of dominance. Analysis of variance showed the importance of both additive and dominance effects in controlling the latent period. Broad-sense heritabilities ranged from 0.91 to 0.98 and narrow-sense heritabilities ranged from 0.59 to 0.92. Significant additive genetic component and moderate narrow-sense heritability indicated the possibility of improving for the longer latent period of stripe rust in breeding programmes.

Key Words: Diallel analysis; Latent period; *Puccinia striiformis*; Stripe (yellow) rust; Wheat

INTRODUCTION

Stripe rust (yellow rust), caused by *Puccinia striiformis* West.f.sp. *tritici*, is an important disease of wheat (*Triticum aestivum* L.) in many parts of the world. This disease can cause significant losses to wheat production. Frequent stripe rust epidemics have been reported in Iran (Bamdadian, 1984). Non-durability of resistance in vulnerable cultivars that contain only specific major genes for resistance have caused breeders have turned their attention to adult-plant resistance or slow rusting. Slow rusting resistance in wheat, inhibits, the development of yellow rust, resulting in a longer latent period, smaller and fewer pustules per square unit of leaf area (Ohm & Shaner, 1976). The effect of these components of slow rusting accumulates over several infection cycles of the pathogen and results in a slower development of the disease in the field (Shaner & Hess, 1978). Stripe rust resistance is controlled by major (Gerechter-Amitai *et al.,* 1974; Gerechter-Amitai *et al.,* 1989), minor (Gerechter-Amitai *et al.,* 1974; Reinhold *et al.,* 1983) or temperature-sensitive genes (Gerechter-Amitai *et al.,* 1981; Gerechter-Amitai *et al.,* 1989). The latent period is one of the most important components of slow rusting resistance (Parlevliet, 1975; Ghannadha *et al.,* 1995) and the easiest component to analyse (Shaner, 1980; Shaner & Finney, 1980) since it can be measured with least error (Kuhn *et al.,* 1978; Shaner *et al.,* 1978; Shaner & Finney, 1980). The variation for the latent period has been reported for stripe rust by Cromey (1992), Ghannadha *et al.* (1995) and Dehghani *et al.* (2002). The object of the current study was to determine the genetic control of the longer latent period of stripe rust resistance in five wheat cultivars by estimating components of genetic variance using the diallel method.

MATERIALS AND METHODS

Five wheat genotypes with different resistance levels to *P. striiformis* (Bolani, Brock, Domino, Elit-Lep, and Kotare) were intercrossed in a half-diallel mating system, to obtain 10 hybrid combinations. Yellow rust samples were collected from different locations of Iran. Pathotype nomenclature follows the system described by Johnson et al. (1972), using the suffix introduced by Wellings and McIntosh (1990). Four pathotypes, 134E134A+, 140E72A+, 174E174A+ and 230E15A+, were selected for use in the greenhouse. Urediniospores of each pathotype were multiplied on the susceptible cultivar, Bolani, in the greenhouse. Each day, inoculum was collected, partially dried, and then sealed in plastic-lined aluminum foil bags and stored in freezer at 80°C. Before use, inoculum of each pathotype was heat-shocked by immersing in warm water (42°C) for 4 min, (Ghannadha *et al.,* 1995). The parents and F_1 progenies were planted in 10-cm pots, in each of four separate experiments for each pathotype. The pots were placed in the greenhouse 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 liter and were then left in a darkened dew chamber for 24 h at 10°C to encourage urediniospore germination, penetration and infection. The seedlings were transferred to the greenhouse and maintained in a light: dark period of 16.00: 8.00 h with a light intensity of 16000 LX and temperature of 15/10°C (Ghannadha *et al.*, 1995; Dehghani & Moghaddam, 2004). 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. Daily assessments continued until 20th day. An experiment for each pathotype was conducted using randomized complete block design with three replications. 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 (Hayman, 1954; Mather & Jinks, 1982) were also estimated. In the second case, the data were analyzed to estimate general and specific combining ability effects (GCA & SCA, respectively, Griffing, 1956).

RESULTS

Analysis of variance showed significant differences among treatments for the latent period of all pathotypes (data not shown). The latent period of the pathotypes under study ranged from 7.42 days to 15.07 days for Domino (Table I). All the diallel statistics for the latent period for the four pathotypes are presented in Table II. With the exception of pathotype 174E174A+ that W_r–V_r value and regression coefficient were significant for it, for the rest of pathotypes W_r–V_r value and regression coefficient were not significant, and this confirms lack of epistasis for latent period in this study. Additive genetic variance (D) was greater than the dominance genetic variance (H_1 and H_2) for pathotypes 140E72A+ and 230E15A+; whereas, for pathotypes 174E174A+ and 134E134A+ dominance variance (H_1 and H_2) were greater. Estimates of F were positive for all pathotypes, indicating the inequality of gene frequencies with an excess of dominant over recessive alleles. The degree of dominance, $(H_1/D)^{1/2}$, indicated full dominance for pathotype 174E174A+ while it was partial dominance for other pathotypes. Existence of dominance was also confirmed by W_r+V_r values. The gene frequencies indicated the inequality of genes for increasing and decreasing the latent period, except for the pathotype 230E15A+. The proportion of dominant to recessive alleles for all parents indicated dominant alleles were more. Narrow-sense heritability values (0.59-0.92) were less than broad-sense (0.91-0.98). The graphic analysis (Fig. 1) for pathotype 134E134A+ shows that Bolani contained most recessive alleles, while Elit-Lep contained most dominant alleles. For pathotype 174E174A+, Brock contained most recessive alleles, while Kotare contained most dominant alleles. For pathotypes 140E72A+ and 230E15A+, Domino contained most recessive alleles and Elit-Lep contained most dominant alleles. The intercept of the regression line for all pathotypes was above the origin, indicating partial dominance. Both GCA and SCA were highly significant (Table III).The ratio $2MS_{GCA}/(2MS_{GCA}+MS_{SCA})$,indicating the relative importance of additive to nonadditive effects. Using Griffing's method narrow-sense and broad-sense heritabilities ranged from 0.70 to 0.96 for all pathotypes. Estimates of GCA and SCA effects for crosses are given in Table 4. For the pathotypes 174E174A+ and 140E72A+, the GCA for Kotare and Domino was positive, for the pathotypes 134E134A+ and 230E15A+, the GCA for Elit-Lep and Brock was positive, suggesting they are suitable parents for obtaining a longer latent period. The highest SCA was observed in the crosses of Domino×Kotare for pathotypes 134E134A+ and 230E15A+, Bolani×Brock for pathotype 140E72A+ and Elit-Lep×Brock for pathotype 174E174A+, suggesting the presence of dominance for the longer latent period in these hybrids.

Fig. 1. The W_r, V_r graphs of latent period from crosses of five cultivars of wheat (1, Bolani; 2, Brock; 3, Kotare; 4, Domino and 5, Elit-lep) in response to four pathotypes of stripe rust

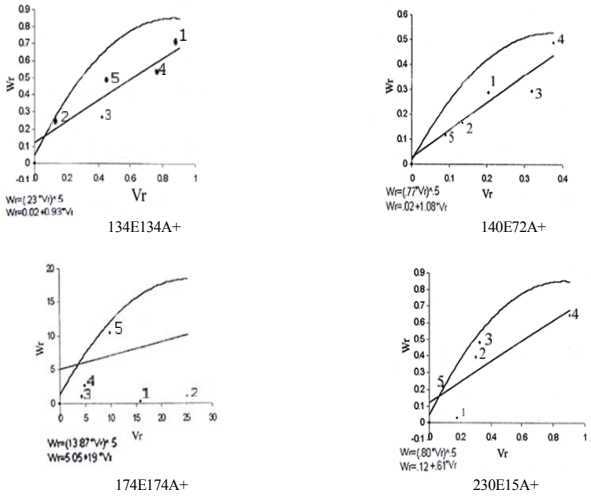

Table I. Duncan's multiple ranges of five cultivars and their progenies for the latent period in response to four pathotypes of stripe rust

Cultivar	Pathotype			
	134E134A+	140E72A+	230E15A+	174E174A+
Bolani	8.077abcd	11.87cde	13.87b	11.33ef
Bolani×Brock	7.97cd	12.13bcd	12.47cd	11.80def
Bolani×Domino	8.157abcd	11.80cde	12.07d	11f
Bolani×Elit-Lep	8.50a	11.73cde	13.00bcd	11.53ef
Bolani×Kotare	8.17abcd	11.20e	12.93bcd	11.80def
Brock	7.94cd	11.73cde	12.80bcd	12.27cde
Brock×Domino	8.11abcd	11.80cde	13.60bc	12.80bcd
Brock×Elit-Lep	7.81de	12.40abc	12.93bcd	11.60ef
Brock×Kotare	8.303abc	11.07e	12.73bcd	11.47ef
Domino	7.42e	13.20a	15.07a	13.80a
Domino×Elit-lep	8.46ab	11.20e	13.20bcd	11.87def
Domino×Kotare	7.92cd	11.40de	13.47bc	11.93def
Elit-Lep	7.43e	12.40abc	13.53bc	13.33ab
Elit-Lep×Kotare	7.93cd	12.80ab	13.87b	13.20abc
Kotare	8.033bcd	11.87cde	13.40bcd	11.33ef

Values followed by the same letter do not differ significantly at the 0.05 level (Duncan's multiple range test).

Table II. Genetic parameter for the latent period of stripe rust in five cultivars of wheat

Parameter	Pathotype			
	134E134A+	140E72A+	230E15A+	174E174A+
$W_r + V_r$	0.007*	0.257*	1.245	357.821*
$W_r - V_r$	0.002	0.017	0.103	508.049*
$\beta - 1$	0.023	0.026	0.012	5.05
D	0.21±1.11*	0.69±0.04*	0.62±0.15*	10.79±8.74
H_1	9.97±3.00*	0.38±0.11*	0.41±0.41	43.34±23.62
H_2	7.28±2.72*	0.23±0.10*	0.339±0.37	28.70±21.43
F	0.13±2.7*	0.37±0.10*	0.009±0.38	13.35±21.85
$(H_1/D)^{1/2}$	0.680	0.740	0.811	2.004
Dom. /rec. genes	1.096	2.131	1.018	1.893
h^2_{BS}[a]	0.87	0.87	0.70	0.81
h^2_{NS}[b]	0.57	0.62	0.56	0.37

* $P < 0.05$

[a,b] Broad- and narrow-sense heritability, respectively

Table III. Mean squares of general and specific combining ability for the latent period of four pathotypes of stripe rust

S.O.V.	d*f*	Pathotype			
		134E134A+	140E72A+	230E15A+	174E174A+
GCA[a]	4	0.241**	0.945**	1.289**	17.686**
SCA[b]	10	0.036*	0.128*	0.231*	11.237*
Error	28	0.019	0.077	0.180	3.243
Ratio[c]		0.930	0.936	0.917	0.763
h^2_{BS}		0.964	0.963	0.939	0.934
h^2_{NS}		0.897	0.902	0.862	0.709

** $P < 0.01$

[a] General combining ability

[b] Specific combining ability

[c] $2MS_{GCA} / (2MS_{GCA} + MS_{SCA})$(Baker 1978)

DISCUSSION

Amongst all mating designs, diallel matings, especially half diallel (Kearsey, 1965) provide a simple and convenient method for estimating genetic parameters. The efficiency of selection depends on the amount of genetic variability in the population under study and the heritability of the character concerned. Further partitioning of genetic variance into its components by methods such as the diallel design, measure the type of the gene action involved in the expression of traits (Mather & Jinks, 1982). Information about the gene action in the latent period of stripe rust helps in deciding the type of the breeding procedure for the genetic improvement of slow rusting wheat (Dehghani & Moghaddam, 2004). The results indicated, there were genetic differences among genotypes and also reversal of dominance for the latent period of different pathotypes. The broad-sense heritability estimates were high, indicating that environmental effects were quiet small on the expression of the latent period (Roy, 2000). Ghannadha *et al.* (1995) and Dehghani and Moghaddam (2004) reported similar results for the latent period of stripe rust. Narrow-sense heritability estimates, however, were mostly high or moderately high indicating that selection for the longer latent period may be effective. A range of 23-92% for heritability of slow rusting in small grain has reported in the literature (Lee & Shaner, 1985a). In the present experiments, genetic diversity among parents was demonstrated by the scatter of the parental array points along the regression line of the Wr/Vr analysis. Hence, array point near the origin represents the array whose common parent contains most of the dominant and faraway from the origin refers to the array whose common parent possesses most of the recessive genes. Points between these extremes represent those arrays possess different proportions of dominant and recessive genes (Mather & Jinks, 1982). The results of study suggested that parents having a lower phenotypic mean (short latent period) were dominant when compared with those having a higher phenotypic mean (Mather & Jinks, 1982). Therefore, varieties such as Brock for 174E174A+ pathotype, Domino for 140E72A+ and 230E15A+ pathotypes possess most of recessive alleles for the longer latent period and could be used in breeding programmes involving these pathotypes. The results of this investigation suggest that the latent period of stripe rust in genetically controlled. Also, both dominance and additive gene action were involved in the genetic control of the latent period. 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. This suggests that the possibility of the selection for the longer latent period in breeding

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

134E134A+	**Bolani**	**Brock**	**Kotare**	**Domino**	**Elit-Lep**
Bolani	0.18*	0.16*	-0.15*	-0.063	0.21*
Brock		0.11*	0.033	0.13*	0.055
Kotare			-0.23*	0.31*	0.046
Domino				-0.16*	0.13*
Elit-Lep	SE_{GCA}= 0.046	SE_{SCA} = 0.095			-0.093*
140E72A+	**Bolani**	**Brock**	**Kotare**	**Domino**	**Elit-Lep**
Bolani	-0.139*	0.429*	0.39*	0.143	0.181
Brock		-0.396*	0.019*	-0.219	-0.095
Kotare			0.242*	0.143	-0.533*
Domino				0.509*	-0.333*
Elit-Lep	SE_{GCA}= 0.094	SE_{SCA} = 0.191			0.215
230E15A+	**Bolani**	**Brock**	**Kotare**	**Domino**	**Elit-Lep**
Bolani	-0.39*	-0.39*	-0.12	-1.03*	0.098
Brock		0.40*	-0.17	0.1	0.04
Kotare			0.12	0.18	0.05
Domino				0.63*	-0.033*
Elit-Lep	SE_{GCA}= 0.14	SE_{SCA} = 0.29			0.03
174E174A+	**Bolani**	**Brock**	**Kotare**	**Domino**	**Elit-Lep**
Bolani	-1.75*	-7.45*	-1.78*	-1.84*	0.96
Brock		-0.12	0.57	1.18	2.93*
Kotare			1.48*	-0.34	1.46*
Domino				1.73	0.6
Elit-Lep	SE_{GCA}= 0.60	SE_{SCA} = 1.24			-1.33*

programmes (Ghannadha *et al.* 1995; Dehghani & Moghaddam, 2004).

REFERENCES

Baker, R.J., 1978. Issues in diallel analysis. *Crop Sci.*, 18: 533–6

Bamdadian, A., 1984. Variation in pathogenicity and evolution of *puccinia striiformis* West. in Iran during 1980–84. *In: Proc. European Mediterranean Cereal Rusts Conference,* pp. 219–22

Cromey, M.G., 1992. Adult plant resistance to stripe rust (*puccinia striiformis*) in some New Zealand wheat cultivars. *N Z J. Crop Hort. Sci.*, 20: 413–9

Dehghani, H., M. Moghaddam, M.R. Ghannadha, M. Valizadeh and M. Torabi, 2002. Inheritance of the latent period of stripe rust in wheat. *J. Genet. Breed.,* 56: 155–63

Dehghani, H. and M. Moghaddam, 2004.Genetic analysis of the latent period of stripe rust in wheat seedlings. *J. Phytopathol.,* 152: 325–30

Gerechter–Amitai, Z.K. and A. Groma, 1974. Inheritance of resistance to stripe rust (*Puccinia striiformis*) in crosses between wild emmer (*Triticum dicoccoides*) and cultivated tetraploid and hexaploid wheats. 1. *Triticum durum*. *Euphytica.*, 23: 387–92

Gerechter–Amitai, Z.K., E.L. Sharp and M. Reinhold, 1981. Temperature sensitive genes for stripe rust resistance in *Triticum dicoccoides* indigenous to Israel. *(Abstr.) Phytopathol.,* 71: 218

Gerechter–Amitai, Z.K. and C.H. Van Silfhout, 1989. Race–specificity of temperature–sensitive genes for resistance to *Puccinia striiformis* in *Triticum dicoccoides*. *Euphytica.*, 43: 7–14

Ghannadha, M.R., I.L. Gordon and M.G. Cromey, 1995. Diallel analysis of the latent period of stripe rust in wheat. *Theor. Appl. Genet.*, 90: 471–6

Griffing, B., 1956.Concept of general and specific combining ability in relation to diallel crossing systems. *Australian J. Biol. Sci.*, 9: 463–93

Hayman, B.I., 1954. The theory and analysis of diallel crosses. *Genetics.*, 39: 798–805

Johnson, R., R.W. Stubbs, E. Fuchs and N.H. Chamberlain, 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. *Trans. Br. Mycol. Soc.*, 58: 475–80

Kearsey, M.J., 1965. Biometrical analysis of a random mating population: a comparison of five experimental designs. *Heredity,* 20: 205–35

Kuhn, R.C., H.W. Ohm and G. Shaner, 1978. Slow leaf rusting resistance in wheat against twenty–two isolates of *Puccinia recondita*. *Phytopathol.,* 68: 651–6

Lee, T.S., and G. Shaner. 1985a. Oligogenic inheritance of length of latent period in six slow leaf–rusting wheat cultivars. *Phytopathol.,* 75: 636–43

Mather, K. and J.L. Jinks, 1982. *Biometrical Genetics. The Study of Continuous Variation*, 3rd edition, p. 396. Chapman and Hall, London, New York

McIntosh, R.A. and C.R. Wellings, 1986. The Daniel McAlpine memorial lecture. Wheat rust resistance–the continuing challenge. *Australian Plant Pathol.*, 15: 1–8

Ohm, H.W. and G.E. Shaner, 1975. Segregation for slow leaf–rusting in wheat. *Am. Soc. Agron. Abstr.*, p. 59

Parlevliet, J.E., 1975. Partial resistance of barley to leaf rust, *Puccinia hordei*. 1. Effect of cultivar and development stage on latent period. *Euphytica*, 24: 21–7

Roy, D., 2000. *Plant Breeding Analysis and Exploitation of Variation,* p. 701. Alpha Science International Ltd, India

Reinhold, M., E.L. Sharp and Z.K. Gerechter–Amitai, 1983. Transfer of additive "minor–effect" genes for resistance to *Puccinia striiformis* from *Triticum dicoccoides* into *Triticum durum* and *Triticum aestivum. Canadian J. Bot.*, 61: 2702–7

Shaner, G., 1980. Probits for analyzing latent data in studies of slow rusting resistance. *Phytopathol.,* 70: 1179–82

Shaner, G. and F.D. Hess, 1978. Equations for integrating components of slow leaf–rusting resistance in wheat. *Phytopathol.,* 68: 1464–9

Shaner, G., H.W. Ohm and R.E. Finney, 1978. Response of susceptible and slow rusting wheats to infection by *Puccinia recondita*. *Phytopathol.,* 68: 471–5

Shaner, G. and R.E. Finney, 1980. New sources of slow rusting resistance in wheat. *Phytopathol.,* 70: 1183–86

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