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GENETICS OF SLOW LEAF RUSTING IN WHEAT

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ABSTRACT

Two sets of materials were produced involving 22 wheat cultivars to study the genetics of slow leaf rusting. In Set I, seven generations (two parents, F_1, F_2, F_3, BC_1 and BC_2) of three crosses (Kalyan Sona x Pavon, Kalyan Sona x WH 147, and K 68 x Veery 'S') were raised and the data analysed to estimate various genetic parameters and test the adequacy of models. In Set II, 60 triple test-cross families produced by crossing 20 wheat cultivars with three testers (Kalyan Sona, Pavon and their F_1) were evaluated to detect epistasis and estimate D and H components of genetic variation. The 6-parameter model explained best the inheritance of slow leaf rusting in the material of Set I. Partial dominance was indicated by all the three crosses and the genes causing less disease were dominant over their alleles causing more disease. Duplicate epistasis was present in all the three crosses. The results of the two sets were similar except that a case of complete dominance tending towards overdominance was indicated in Set II.

Key words: Slow leaf rusting, wheat, genetic parameters.

Among the various diseases of wheat, the rusts cause maximum damage and among rusts brown rust is the most serious disease in the Indian subcontinent. The development of completely brown rust resistant varieties of wheat does not seem to play a very important role in the long run because of the appearance of new races of the pathogen causing this rust. The ideal way of controlling such plant diseases would be the development of varieties with durable resistance. A genotype that can reduce the rate of disease development ultimately suffers less damage. Though the information on genetics of slow leaf rusting in wheat is scanty, most of the reports available on this aspect indicate that slow leaf rusting is a quantitative trait and is mainly controlled by additive gene effects [1–3]. Therefore, an effort has been made to study the genetics of slow leaf rusting in wheat by estimating different biometrical parameters.

MATERIALS AND METHODS

Two sets of materials were produced involving 22 wheat cultivars, namely, WH 147, WH 157, WH 283, WH 291, WH 416, WL 711, HD 2009, HD 2285, K 68, S 308, Kalyan Sona,

Pavon, Torim 73, India 66, Veery 'S', Tan 'S', Dove 'S', Siete Cerros 66, PVI/NAC, KA 'S'/NAC, Jupateco 76, and Baya 'S'. In Set I, three crosses (Kalyan Sona x Pavon, Kalyan Sona x WH 147 and K 68 x Veery 'S') involving two fast rusting (Kalyan Sona and K 68) and three slow leaf rusting varieties (Pavon, WH 147 and Veery 'S') were made. Seven generations (both parents, F_1, F_2, BC_1, BC_2 and F_3) of each cross were grown together in 2.5 m long rows spaced at 30 cm in randomized block design with three replications. The nonsegregating populations (parents and F_1) were raised in 2-row plots, BC_1, BC_2 and F_3 families in 4 row plots, and F_2s in 6-row plots.

In the Set II, the varieties Kalyan Sona and Pavon and their F_1 were crossed as L_1, L_2 and L_3 testers, respectively, with each of the remaining 20 cultivars to produce 60 triple test cross (TTC) families. Each TTC progeny and the 22 parents were grown in single-row plots of 2.5 m length, spaced 30 cm apart in randomized block design with three replications.

The experimental material in the field was surrounded by infector rows of highly susceptible varieties (Agra Local and Kalyan Sona). These infector rows were inoculated with the mixture of the races of brown rust prevalent in India late in the evenings and heavy irrigation was applied to maintain high humidity in the field. Rust intensities based on the modified Cobb's scale of cereal rust score were recorded on five random plants from P_1, P_2 and F_1, 50 plants from BC_1, $BC_2$and F_3, and 90 plants from F_2 in each replication in Set I, and on 5 random plants in each treatments of Set II on five different days at 7-day intervals, starting from 13 March 1989 in Set I and 16 March 1989 in Set II. The area under disease progress curve (AUDPC) was computed for each plant after changing the scale of rust intensity data using arcsin transformation.

The transformed data of Set I were analysed as per the method of [4] described by [5]. The data of Set II were analysed according to [6]. Epistasis was tested against replicate error (40 d.f.) if the replicate error was significant against within family error (720 d.f.). The two components of epistasis, i and j & l, were tested against their respective replicate interactions (calculated for 2 d.f. and 38 d.f., respectively). Similarly, sums and differences were tested against their replicate interactions (38 d.f.) if these interactions were significant against their corresponding within families errors (720 d.f. and 480 d.f., respectively).

RESULTS AND DISCUSSION

The fitting of genetic models of increasing complexity to the means of seven generations (Set I) for slow leaf rusting as quantified by area under disease progress curve (AUDPC) indicated that not only the additive–dominance model, but also the models which allow homozygote x homozygote (i) and homozygote x heterozygote (j) interactions were inadequate to explain the genetic basis of slow leaf rusting in all the three crosses (Table 1). However, the 6-parameter model allowing all three kinds of epistasis (additive x additive,

Table 1. Test of goodness of fit of different genetic models and estimates of genetic parameters for slow leaf rusting in three wheat crosses

Cross and model	χ^2	Estimate ± standard error					
		m	(d)	(h)	(i)	(j)	(l)
Cross: Kalyan sona x Pavon							
m (d) (h)	81.6	—	—	—	—	—	—
m (d) (h) (i)	37.1	—	—	—	—	—	—
m (d) (h) (i) (j)	36.1	—	—	—	—	—	—
m (d) (h) (i) (j) (l)	0.2	3.95 ± 0.23	4.69 ± 0.08	−3.29 ± 0.37	1.64 ± 0.25	−0.69 ± 0.34	1.55 ± 0.26
Cross: K 68 x Veery 'S'							
m (d) (h)	166.3	—	—	—	—	—	—
m (d) (h) (i)	82.8	—	—	—	—	—	—
m (d) (h) (i) (j)	52.6	—	—	—	—	—	—
m (d) (h) (i) (j) (l)	2.5	3.46 ± 0.23	5.71 ± 0.07	−4.71 ± 0.39	2.33 ± 0.24	−3.11 ± 0.35	1.98 ± 0.28
Cross: Kalyan sona x WH 147							
m (d) (h)	80.7	—	—	—	—	—	—
m (d) (h) (i)	38.9	—	—	—	—	—	—
m (d) (h) (i) (j)	37.7	—	—	—	—	—	—
m (d) (h) (i) (j) (l)	0.3	3.82 ± 0.24	4.82 ± 0.09	−3.37 ± 0.38	1.70 ± 0.26	−0.70 ± 0.35	1.61 ± 0.26

additive x dominance and dominance x dominance) was adequate; thus additive and dominance gene effects in association with digenic interactions explained the inheritance of slow leaf rusting best in the present material. The estimates of all the six parameters were highly significant in all three crosses except the parameter (j) which showed borderline significance in the crosses Kalyan Sona x Pavon and Kalyan Sona x WH 147. This indicates that (j) type epistasis was relatively less important in these two crosses than the other two types of interactions which showed almost equal importance in all the three crosses. However, in a highly self-fertilized crop like wheat, (i) component of epistasis can be exploited easily being fixable like the additive component. On the other hand, the unfixable components of epistasis, (j) and (l), do not seem to have any advantage in wheat breeding unless hybrid wheat becomes a commercial proposition in the near future. The magnitude of (d) was relatively higher than that of (h) in all three crosses indicating greater importance of additive gene effects in the control of this character than the dominance gene effects. The estimates of (h) in all the three crosses were negative, indicating that the genes determining less leaf rust were, in general, dominant over the alleles causing more disease. Further, since estimates of (h) and (l) in all the three crosses had different signs, there was evidence of duplicate epistasis for leaf rust in the present material. This type of epistasis cannot be easily exploited in crop improvement since in this case the increase in one parameter will automatically be followed by decrease in the other.

The results of Set II (Table 2) show that epistasis and its i and j & l subcomponents as well as sums and differences were highly significant when tested against their respective replicate interactions (since all these interactions were significant against their corresponding within families errors). Thus, all three components of genetic variation (additive, dominance and epistatic) were responsible for the control of leaf rust in this material. The estimates of D and H components were almost equal, the estimate of H being slightly higher than that of D. The degree of dominance was thus slightly higher than 1. These results confirm the results of Set I. The only discrepancy between the results of the two sets was that Set I gave an indication of partial dominance in all the three crosses, whereas Set II was a case of full dominance. This discrepancy may be attributed to the cancelling effect of genes in Set I. Many research workers have reported the involvement of all the three kinds of gene effects in the control of leaf rust in wheat [2, 7]. However, some reports [2, 3, 8] on the genetics of slow leaf rusting indicate that this trait is under the control of additive gene effects.

Table 2. Analysis of variance for epistasis, sums and differences and estimates of additive (D) and dominance (H) components and degree of dominance for slow leaf rusting in triple test-cross families of wheat

Item/Parameter	d.f.	Mean squares
Epistasis : $(\bar{L}_{1i} + \bar{L}_{2i} - 2\,\bar{L}_{3i})$		2019.8
i type	1	41.1
j & l type	19	18.7
Sums : $(\bar{L}_{1i} + \bar{L}_{2i} + \bar{L}_{3i})$	19	11.7
Difference : $(\bar{L}_{1i} - \bar{L}_{2i})$	19	9.2
D	—	1.0
H	—	1.2
$\sqrt{H/D}$	—	1.1

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