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Inheritance of adult field resistance to yellow rust disease among broad-based hexaploid spring wheat germplasm

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Abstract Complete F_1 and F_2 diallel crosses were used to investigate the inheritance of yellow rust resistance among eight bread wheat lines, developed by CIMMYT for the East African Highlands, which showed a wide response to this disease. Both diallel sets were grown at a site with a high incidence of yellow rust, although for one season, during which the F_1 diallel was grown, disease incidence was unusually low. Analyses disclosed the presence of additive, dominance and epistatic effects among those genes controlling rust resistance, with the former being the most important. At normal disease levels, excluding two arrays having resistant common parents removed non-allelic interactions from the F_1 diallels. For all F_2 diallels, and the remaining F_1 diallel, omitting two arrays based on susceptible parents removed these interactions. Local selection of material from a broadly based germplasm appears to be a feasible method of developing adapted cultivars resistant to endemic diseases.

Key words Additivity · Diallel cross · Dominance · Epistasis · Wheat · Yellow rust

Introduction

Yellow rust, caused by *Puccinia striiformis* Westend., is a serious disease of wheat (*Triticum aestivum* L.) in areas where cool, moist weather prevails, as in northwestern Europe and the mountainous regions of South America and East Africa (Stubbs 1988). In Uganda, it is one of the main biotic factors limiting wheat production. Although increased yields as a result of chemical control have been reported in several European countries (Buchenauer 1982), for the small-scale farmer in a developing country, where resources are always limited, the wholesale use of fungicides cannot be recommended. Genetic resistance is the most economical and environmentally safe control measure (Chen and Line 1992).

The Uganda Wheat Development Project (UWDP) has identified several lines adapted to local environments from germplasm developed by the International Maize and Wheat Improvement Centre (CIMMYT). Although CIMMYT has analysed selected groups of genotypes, it is recognized that this work should be expanded (Rajaram et al. 1988). Yellow rust is the most environmentally sensitive of the rusts (Röbbelen and Sharp 1978), a characteristic that creates problems when assessing host plant resistance. Moreover, resistance genes also appear to be sensitive to the environment. These findings may explain the conflicting reports on just how resistance to yellow rust is inherited in wheat (for a review see Röbbelen and Sharp 1978). Thus, recessive and dominant monogenic resistance, as well as resistance controlled by minor genes, have been reported (Stubbs 1985).

The genetic basis of adult field resistance to yellow rust among wheat cultivars adapted to Ugandan environments is not known. In the investigation presented here information was sought on the inheritance of resistance to this disease, which should enable a more comprehensive breeding strategy to be developed.

Materials and methods

Eight bread wheat lines from the UWDP were chosen for this investigation. The lines were selected at Kalengyere, a location with a high incidence of yellow rust, where they exhibited a wide response

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Table 1 Designation, parentage, pedigree and rust response of the parents

Code name/pedigree	Source[a]	Yellow rust reaction
1 BURI CM58340-A-1Y-3Y-2M-2Y-0M	2nd HRWSN	Resistant
2 KENYA CHIRIKU K. TEMBO/CARPINTERO"S"	NPBRC	Resistant
3 ESDA/LIRA CM78428-017M-013M-013Y-03AL-3Y-3AL-0Y	2nd HRWSN	Resistant
4 VEE"S"/JUP73/EMU"S"//GJO"S" CM74465-05AP-300AP-4AP-300AL-0AP	RBWONLR	Moderately resistant
5 ATTILA CM85836-4Y-0M-0Y-OPZ	4th HRWSN	Moderately susceptible
6 CY8801	5th HCWSN	Susceptible
7 F60314.76/4/CNO76/7C//KAL/BB/3/PC1"S"/5/CNO79	13th SNACWYT	Susceptible
8 CAR853/COC//VEE"S"/3/E7408/PAM"S"/HORK"S"/PF73226	13th SNACWYT	Susceptible

[a] HRWSN, High rainfall wheat screening nursery, CIMMYT, Mexico; NPBRC, National Plant Breeding Research Centre, Njoro, Kenya; RBWONLR, Regional Bread Wheat Observation Nursery for Leaf Rust, ICARDA, Syria; HCWSN, Hot Climate Wheat Screening Nursery, CIMMYT, Thailand; SNACWYT, Screening Nursery for African Cooperative Wheat Yield Trial, CIMMYT, East Africa

to this disease (Table 1). For this investigation the parents were intercrossed and selfed in a complete diallel crossing scheme, giving rise to 56 F_1 hybrids and F_2 populations derived by selfing the F_1s. Between 1994 and 1996 F_1 and F_2 diallels were each grown in three consecutive seasons at Kalengyere, located 2400 metres above sea level in the south-western highlands of Uganda. During the trial period average daily temperature was 16°C. Precipitation was bimodal, with relatively low rainfall (480 mm) in season (A), lasting from March to August, and high rainfall (750 mm) during season (B), from September to March. A randomized, complete block design with two replicates was used throughout. For the F_1s experimental plots comprised two rows of 1.5-m length and 0.3-m inter-row spacing, whereas for the F_2s the two experimental rows were 5 m long. Spacing between plants was 0.15 m. Nitrogen was applied at planting at a rate of 50 kg ha^{-1}.

Yellow rust severity was scored on the flag leaf of individual plants when the rust on the most susceptible parent was about 100%, and most of the leaf surface was covered with uredinia. Disease severity was recorded between the late milk and early dough stages (Zadok's growth stages 77–83) using the modified Cobb scale to estimate the percentage of possible tissue rusted (Peterson et al. 1948). Host response to infection was scored using T (= 0.1) to indicate immunity; R (= 0.2) to indicate resistance in plants showing miniature uredinia; MR (= 0.4) to indicate moderate resistance in plants exhibiting small uredinia; MS (= 0.8) to indicate moderate susceptibility in plants with moderate-sized uredinia (smaller than fully susceptible type); S (= 1) for complete susceptibility (Wagoire 1997). The disease severity and host response scores were multiplied together to give the coefficient of infection for data analysis.

Analyses of variance were carried out on plot means using the method developed by Hayman (1954) and described in detail by Mather and Jinks (1982) and Hill et al. (1998). Genetic analyses, based on an analysis of array variances (Vr_i) and covariances (Wr_i), followed the procedures developed by Jinks (1954). Adequacy of the additive-dominance model of gene action was tested by a joint regression analysis of Wr_i on Vr_i and analyses of the ($Wr_i + Vr_i$) and ($Wr_i - Vr_i$) (Mather and Jinks 1982; Hill et al. 1998).

Results and discussion

Host response for each cross and season, averaged over replicates, is shown for the F_1 diallel in Table 2, from which it is apparent that cultivar 3, 'Esda/Lira', is highly resistant to yellow rust, followed by 'Kenya Chiriku' and 'Buri'. It should be noted that disease incidence in the 1994B season was unusually low. Comparable results were obtained from the F_2 data (not shown), though seasonal differences are not so marked. Nevertheless, overall, F_1 hybrids are more resistant to yellow rust than F_2 populations in both those seasons when they are grown together (1995A and B), though this difference is only significant in 1995A.

Mean squares from the analysis of variance, combined across seasons, are given in Table 3 for both F_1 and F_2 diallels. Although additive and non-additive genetic effects (a and b items) are highly significant, regardless of whether they are tested against the pooled seasonal interactions or their own interaction with seasons, the former is clearly the major contributor to the variability among these lines. In the F_1 diallel, non-additive variation is due to dominance deviations unique to each cross (b_3), that is specific combining ability (s.c.a.) effects, whereas in the F_2 data there is also evidence of directional dominance (b_1) and asymmetrical distribution among the parents of those genes exhibiting dominance for this character (b_2). Although additive effects interact with seasons in both F_1 and F_2 diallels, this is due to changes in the magnitude of the differences between arrays, rather than changes in their ranking, as correlations between general combining ability (g.c.a.) effects across seasons within F_1 and F_2 generations are, with one exception, greater than 0.9. Reciprocal effects (c and d items) are unimportant in these data.

The unusually low disease incidence recorded during the 1994B season is reflected in the analysis of the F_1 data for individual seasons (Table 4). Additive genetic variation is present throughout, but in this particular season non-additive effects are due solely to s.c.a. effects (b_3). For the remaining seasons directional dominance (b_1) and gene asymmetry (b_2) also occur,

Table 2 Mean coeffients of yellow rust infection for the F_1 diallel for the 1994B (upper), 1995A (middle) and 1995B (lower) seasons

Parent	1	2	3	4	5	6	7	8	Array mean
1	0.11	0.29	0.19	0.37	1.19	1.86	7.27	18.02	
	0.25	0.41	0.19	3.75	2.77	3.54	18.86	33.46	
	0.10	0.25	0.00	6.55	11.97	0.00	6.84	28.17	6.10
2	0.29	0.17	0.25	1.06	0.21	1.44	3.44	3.42	
	0.62	0.32	0.10	0.54	0.30	0.87	0.47	6.47	
	0.28	0.03	0.18	0.53	0.47	0.27	0.67	5.89	1.18
3	0.16	0.75	0.11	0.15	0.17	0.45	0.34	0.56	
	0.15	0.14	0.08	0.33	0.29	0.55	0.44	0.49	
	0.38	0.00	0.00	0.00	0.19	1.90	2.16	0.80	0.42
4	0.26	0.53	0.10	1.07	0.23	2.46	3.55	12.33	
	2.80	0.73	0.39	9.00	7.67	25.67	66.34	86.67	
	7.84	1.27	0.09	11.31	15.13	4.66	33.38	66.29	14.99
5	0.22	0.36	0.15	0.16	2.18	3.03	6.52	17.50	
	3.00	0.46	0.23	10.17	16.62	86.67	89.00	95.00	
	2.68	1.21	0.25	25.00	35.15	18.15	31.63	94.29	22.48
6	1.16	1.94	0.84	2.89	4.02	14.99	31.72	43.62	
	4.06	2.45	0.52	24.21	82.50	73.29	97.50	98.87	
	6.34	2.10	2.50	18.49	26.25	51.00	63.14	85.72	30.84
7	2.21	1.04	0.46	12.45	9.27	27.82	19.18	33.15	
	17.04	1.00	1.08	86.84	85.00	89.17	94.17	96.67	
	18.25	4.28	1.32	67.65	68.86	62.95	94.29	96.87	41.29
8	20.18	11.52	1.48	35.38	18.05	34.06	43.15	42.09	
	39.75	0.54	0.46	93.35	93.29	93.35	89.17	94.17	
	21.68	3.85	3.27	81.25	81.25	72.25	99.07	91.25	48.49
Seasonal means									
1994A	7.90								
1995A	30.17								
1995B	23.98								

Table 3 Mean squares from an analysis of variance of the F_1 (1) and F_2 (2) diallels, combined over seasons

Item	*df*	(1)	(2)
Genotypes	63	4 396.09***	4 983.48***
a	7	30 082.75***	36 852.24***
b	28	2 283.59***	1 959.63***
b_1	1	2 258.39	5 184.55***
b_2	7	884.57	454.07*
b_3	20	2 774.50***	2 325.33***
c	7	184.51	47.69
d	21	54.38	37.63
Seasons (S)	2	16 942.49***	4 095.37***
Sxa	14	3 075.07***	399.50**
Sxb	56	447.61	141.14
Sxb_1	2	398.17	619.75*
Sxb_2	14	283.44	95.35
Sxb_3	40	507.54	133.24
Sxc	14	72.56	54.62
Sxd	42	49.04	86.73
Pooled seasonal interactions	126	565.03***	142.10***
Blocks within S	3	173.23*	321.08***
Pooled block interactions	189	54.89	57.83*

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

Table 4 Mean squares from an analysis of variance of the F_1 diallels conducted at Kalengyere

Item	*df*	1994B	1995A	1995B
a	7	1 984.966***	20 355.336***	13 892.580***
b	28	150.350***	1 915.099***	1 113.349***
b_1	1	83.393	618.832***	2 352.499***
b_2	7	21.285	1 034.880***	395.287***
b_3	20	198.871***	2 287.989***	1 302.714***
c	7	48.766	25.004	255.860
d	21	28.076	28.631	95.754
Blocks	1	248.840***	169.720*	101.125
Pooled error	63	30.877	29.927	104.086

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

with F_1 hybrids being on average more resistant to yellow rust than their parents. Additive and non-additive genetic variation exists throughout the F_2 data (Table 5), with F_2 populations also being significantly more resistant to yellow rust than their parents (b_1). Dominance therefore acts towards increased resistance among these bread wheat lines.

A summary of the results from the genetic analyses (Table 6) reveals the widespread contribution which epistasis makes to the non-additive genetic variation (see also Allan et al. 1966; Mohammed et al. 1972; Sharp et al. 1976), reflected both in the departure of the slope of the joint regression line from its expected value of one, and the significance of the arrays item in the $(Wr_i - Vr_i)$ analysis. In the F_1 data its removal again reflects the incidence of yellow rust. Thus, in the 1994B season, when disease incidence was low, excluding arrays 7 and 8, which have the two most susceptible lines as common parent, removes the non-allelic interactions. For the remaining F_1 data, when disease incidence was normal, omitting arrays 1 and 3, based on the two most resistant lines, removes these interactions. In the F_2 diallels, removing arrays 7 and 8 restores an additive-dominance model of gene action.

All the parents grown in these experiments derive, directly or indirectly, from trials conducted by CIMMYT and have, therefore, been bred for wide adaptation, including resistance to various diseases (Rajaram et al. 1996). From these results it is clear that the inheritance of yellow rust resistance in this broadly based material is controlled by genes exhibiting additive, dominance and epistatic effects. Moreover, the different pattern of non-allelic interactions displayed by the F_1s, depending on disease incidence, is evidence of genotype-environment interactions. At first sight, the epistatic effects detected in the 1994B F_1 diallel on the one hand, and the F_2 diallels on the other hand, appear to have similar origins in that they are removed by excluding those arrays based on the most susceptible parents, 7 and 8. But there the similarity ends, however, because only one type of digenic interaction can occur within F_1 families among those genes segregating for yellow rust, namely that between heterozygous pairs of genes. Within the F_2 populations though, homozygous and heterozygous gene combinations can occur among these genes. Potentially, therefore, all three types of digenic interaction could arise. Segregation and recombination of these genes would also explain the different pattern of non-allelic interactions displayed by the F_2 compared to the F_1 diallels grown in 1995 A and B. A particular type of gene action is not, however, the exclusive attribute of a host plant because it also depends on its interaction with a specific pathotype of the fungus. Moreover, the significant additive x seasons interaction (Table 3) indicates that environmental factors are influencing the activity of the fungus, the host or both. Alternatively, different races of the fungus could have occurred in the different seasons. The use of a quantitative, as opposed to a qualitative model of gene action based on arbitrary scales, avoids those problems associated with the artificial grouping of data into discrete classes, which can force an unwarranted genetic architecture onto the data. It is therefore likely that such a model will present a more accurate picture of the response of this broadly based wheat germplasm, developed at CIMMYT and adapted to the East African highlands, to the Ugandan pathotypes of *P. striiformis*.

Table 5 Mean squares from an analysis of variance of the F_2 diallels conducted at Kalengyere

Item	*df*	1995A	1995B	1996A
a	7	14 685.636***	12 283.627***	10 609.873***
b	28	501.992***	803.029***	945.903***
b_1	1	347.989***	1 476.060***	4 600.261***
b_2	7	142.347***	316.409***	186.013
b_3	20	635.581***	939.695***	1 029.147***
c	7	30.699	79.787	46.447
d	21	35.560	99.335	76.189
Blocks	1	86.486	59.610	817.130***
Pooled error	63	32.196	62.606	78.682

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

Table 6 Genetic analysis of yellow rust severity at Kalengyere for (1) the complete 8×8 and (2) the reduced 6×6 F_1 and F_2 diallels (*ns* nonsignificant)

	F_1[a]			F_2[b]		
	1994B	1995A	1995B	1995A	1995B	1996A
(1)						
Joint regression coefficient	0.733 ± 0.064	0.775 ± 0.072	0.837 ± 0.069	0.891 ± 0.045	0.856 ± 0.076	0.968 ± 0.063
Significance of arrays in $(Wr_i + Vr_i)$ analysis	ns	***	***	***	***	***
Significance of arrays in $(Wr_i - Vr_i)$ analysis	*	***	*	**	*	***
(2)						
Joint regression coefficient	0.981 ± 0.051	0.923 ± 0.111	0.892 ± 0.101	0.920 ± 0.074	0.833 ± 0.114	1.076 ± 0.067
Significance of arrays in $(Wr_i + Vr_i)$ analysis	*	***	*	***	**	*
Significance of arrays in $(Wr_i - Vr_i)$ analysis	ns	ns	ns	ns	ns	ns

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

[a] Reduction to a 6×6 diallel by omitting arrays 7 and 8 in 1994B, and 1 and 3 in 1995 A and B

[b] Reduction to a 6×6 diallel by omitting arrays 7 and 8 in all seasons

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