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INHERITANCE OF RESISTANCE TO LEAF RUST IN A WHEAT-RYE RECOMBINANT 'SELECTION 212'

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ABSTRACT

Inheritance of resistance to four pathotypes (77-1, 77-3, 77-4 and 77-5) in Selection 212, a 42 chromosome wheat line developed through homoeologous recombination between wheat and rye chromosomes using monosomic 5B of variety Chinese Spring was studied. A recessive seedling resistance gene effective against each pathotype has been determined based on segregation pattern observed in F_2, F_3, the BC_1F_1 and BC_1F_2 generations of the crosses of Selection 212 with two susceptible lines Agra Local and Chinese Spring. Joint segregation on the F_3 and the BC_1F_2 progenies from the crosses revealed that the same resistance gene is controlling resistance to all the four pathotypes. The adult plant tests suggested the presence of an additional adult plant resistance gene which could have been derived from Chinese Spring, one of the parent of Selection 212.

Key Words : Wheat, rye (*Secale cereale*), leaf rust (*Puccinia recondita*), inheritance, recessive

Rusts are the major diseases of wheat that cause substantial losses to crop yields. Cultivation of resistant varieties is the most economical method in controlling these losses. As the resistance in the cultivars is neutralized with the evolution of new races, breeding for resistance using diverse resistance sources, is the most effective strategy to prolong the life of newly evolved varieties. Diversity for resistance gene pool is enriched by creating new usable variability through the manipulation of chromosome architecture. In this endeavour, rye (*Secale cereale*), a member of tertiary gene pool possessing several desirable traits was exploited to develop a wheat-rye recombinant 'Selection 212' using homoeologous chromosome pairing in the absence of chromosome 5B of *aestivum* wheat [1]. A self compatible amber seeded diploid rye (2n=14) was crossed with monosomic 5B line of hexaploid (2n=42) wheat variety Chinese Spring. A 27 chromosomes F_1 hybrid exhibiting extensive homoeologous pairing was backcrossed to wheat variety Sonalika. In BC_1F_7, a cytologically stable ($2n=42=21^{II}$) line was designated 'Selection 212'. Selection 212 was

reported to be resistant to leaf and stem rusts of wheat [2]. This recombinant was also resistant when tested with 25 pathotypes of *Puccinia recondita* f. sp. *tritici* in seedlings and to most virulent and predominant leaf rust pathotype (77-5) at adult plant stage [3]. The present article reports inheritance of resistance to leaf rust in Sel. 212, against four important pathotypes.

MATERIALS AND METHODS

The F_1, F_2 and F_3 generations from the crosses Selelction 212 × Agra Local (AL), Sel.212 × Chinese Spring (CS) and the reciprocal (CS × Sel. 212) along with BC_1F_1 and BC_1F_2 of the crosses (Selelction 212 × AL) × AL and the BC_1F_2s of (Selelction 212 × AL) × Sel. 212 and (Seelction 212 × CS) × Sel. 212 were tested with pathotypes 77-1, 77-3, 77-4 and 77-5 of *P. recondita tritici* at seedling stage and pathotype 77-5 at adult plant stage. These pathotypes were selected on the basis of their virulence on parental wheat lines of Sel.212 i.e. Sonalika and Chinese Spring and avirulence on parental rye strain, so that the resistance contributed by rye in Sel. 212 can be studied.

All these four pathotypes are virulent on seedlings of lines carrying leaf rust resistance genes *Lr1, Lr2a, Lr2b, Lr2c, Lr3, Lr10, Lr11, Lr12, Lr13, Lr14a, Lr15, Lr16, Lr33, Lr34* and avirulent on *Lr9, Lr19, Lr24, Lr25, Lr28, Lr29* and *Lr32*. These four pathotypes are differentiated on five lines carrying *Lr17, Lr18, Lr20, Lr23* and *Lr26*. Among these resistance genes pathotype 77-1 is avirulent on *Lr17* and *Lr23*, 77-3 on *Lr20* and *Lr23*, 77-4 on *Lr20* and *Lr26* and 77-5 on *Lr18*, and virulent on rest of the lines. The purity of pathotypes was confirmed by testing on differential sets. Parental lines were also included in each set of experiment.

Seedling tests

Seedlings were grown in rectangular trays (11" × 4" × 3"). Ten seeds per row and ten rows in each tray were sown. A week old seedlings were inoculated with urediospore cultures of pathotypes 77-1, 77-3, 77-4 and 77-5 according to the procedure described by Joshi *et al.* [4]. Material was kept in glass house at mean temperature range 13°C to 35°C. Reactions were recorded according to the scale described by Stakman *et al.* [5] after 14 days of inoculation.

Adult plant tests

The material was planted on 15th November in an isolated nursery in field in 1.5 meter long rows with row to row distance of 30 cms and seed to seed distance of 10 cm. The spreader row was planted after every 20th row and also all around the nursery and inoculated with urediospore suspension of pathotype 77-5 in water

Table 1. Seedling and adult plant reactions on parental lines and F_1s involving Selection 212 against pathotypes 77-1, 77-3, 77-4 and 77-5

Parent/Cross	Rust reaction									
	Seedling								Adult plant	
	77-1		77-3		77-4		77-5		77-5	
	Infection type	No. of seedlings	Infection type	No. of seedlings	Infection type	No. of seedlings	Infection type	No. of seedlings	Field response	No. of plants
Selection 212	$X^{=}$ (R)	22	X^{-} (R)	16	X^{-} (R)	13	X^{-} (R)	21	TR(R)	16
Agra Local (AL)	3^{+} (S)	20	3^{+} (S)	14	3^{+} (S)	17	33^{+} (S)	22	90S(S)	11
Chinese Spring (CS)	33^{+} (S)	18	33^{+} (S)	11	33^{+} (S)	14	33^{+} (S)	18	40MR(R)	12
Selection 212 × AL	33^{+} (S)	10	3^{+} (S)	08	3^{+} (S)	09	3^{+} (S)	10	50MR(R)	05
Selection 212 × CS	33^{+} (S)	07	-	-	-	-	33^{+} (S)	09	-	-
CS × Selection 212	33^{+} (S)	05	-	-	-	-	33^{+} (S)	07	-	-

R = Resistant, S = Susceptible, - = Not tested

Table 2. Segregation for seedling and adult plant reaction on F_2s, F_3, BC_1F_1s and BC_1F_2 generations from crosses of Sel. 212 with Agra Local (AL) and Chinese Spring (CS) against pathotypes 77-1, 77-3, 77-4 and 77-5

Population	No. of Seedlings/plants/families				Expected ratio	χ^2	P value
	Total	Res.	Segr.	Susc.			
Seedling reaction to pathotype 77-1							
F_2 (Sel. 212 × AL)	106	27	0	79	1R:3S	0.013	0.95-0.90
F_2 (Sel. 212 × CS)	105	26	0	79	1R:3S	0.003	0.98-0.95
F_2 (CS × Sel. 212)	190	41	0	149	1R:3S	1.186	0.30-0.20
F_3 (Sel. 212 × AL)	80	24	42	14	1R:2Segr:1S	2.700	0.30-0.20
BC_1F_1 [(Sel. 212 × AL) × AL]	65	0	0	65	All S		
BC_1F_2 [(Sel.212 × AL) × AL]	28	0	16	12	1Segr:1S	0.571	0.50-0.30
BC_1F_1 [(Sel.212 × AL) × Sel. 212]	34	16	0	18	1R:1S	0.118	0.80-0.70
BC_1F_1 [(Sel.212 × CS) × Sel. 212]	26	14	0	12	1R:1S	0.154	0.70-0.50
Seedling reaction to pathotype 77-3							
F_2 (Sel. 212 × AL)	75	17	0	58	1R:3S	0.218	0.70-0.50
F_3 (Sel. 212 × AL)	80	23	44	13	1R:2Segr:1S	3.300	0.20-0.10
BC_1F_1 [(Sel.212 × AL) × AL]	36	0	0	36	All S		
BC_1F_2 [(Sel. 212 × AL) × AL]	28	0	14	14	1Segr:1S	0.000	> 0.99
BC_1F_1 [(Sel. 212 × AL) × Sel. 212]	37	16	0	21	1R:1S	0.676	0.50-0.30
BC_1F_1 [(Sel. 212 × CS) × Sel. 212]	46	22	0	24	1R:1S	0.087	0.80-0.70
Seedling reaction to pathotype 77-4							
F_2 (Sel. 212 × AL)	73	16	0	57	1R:3S	0.369	0.70- 0.50
F_3 (Sel. 212 × AL)	80	25	41	14	1R:2Segr:1S	3.075	0.30-0.20
BC_1F_1 [(Sel.212 × AL]	41	0	0	41	All S		
BC_1F_2 [(Sel. 212 × AL) × AL]	28	0	13	15	1Segr:1S	0.143	0.80-0.70
BC_1F_1 [(Sel. 212 × AL) × Sel. 212]	20	09	0	11	1R:1S	0.200	0.70-0.50
BC_1F_1 [(Sel. 212 × CS) × Sel. 212]	17	08	0	09	1R:1S	0.059	0.90-0.80

Population	No. of Seedlings/plants/families				Expected ratio	χ^2	P value
	Total	Res.	Segr.	Susc.			
Seedling reaction to pathotype 77-5							
F_2 (Sel. 212 × AL)	434	110	0	324	1R:3S	0.028	0.90-0.80
F_2 (Sel. 212 × CS)	126	32	0	94	1R:3S	0.011	0.95-0.90
F_2 (CS × Sel. 212)	82	26	0	56	1R:3S	1.968	0.20-0.10
F_3 (Sel. 212 × AL)	80	23	43	14	1R:2Segr:1S	2.475	0.30-0.20
BC_1F_1 [(Sel. 212 × AL) × AL]	59	0	0	59	All S		
BC_1F_1 [(Sel. 212 × AL) × AL]	28	0	14	14	1Segr:1S	0.000	> 0.99
BC_1F_2 [(Sel. 212 × AL) × Sel. 212]	79	42	0	37	1R:1S	0.316	0.70-0.50
BC_1F_1 [(Sel. 212 × CS) × Sel. 212]	84	43	0	41	1R:1S	0.048	0.90-0.80
Adult plant reaction to pathotype 77-5							
F_2 (Sel. 212 × AL)	314	246	0	68	13R:3S	1.741	0.20-0.10
F_3 (Sel. 212 × AL)	77	35	38	04	7R:8Segr:1S	0.738	0.50-0.30
BC_1F_1 [(Sel. 212 × AL) × AL]	29	14	0	15	1R:1S	0.034	0.90-0.80
BC_1F_2 [(Sel. 212 × AL) × AL]	28	0	24	04	3Segr:1S	1.714	0.20-0.10

using hypodermal syringe at boot stage. Three to four tillers in the spreader row were inoculated at intervals of one meter distance. Rust severity was recorded according to the scale described by Peterson *et al.* [6].

Joint segregation tests

The joint segregation test [7] between the genes effective against any two pathotypes was carried out by setting the distribution frequency of the F_3 and the BC_1F_2 families in a two way contigent table. Chi-square values were first calculated for deviation due to genes giving resistance to two pathotypes individually and then deviation due to combined segregation for resistance genes. Chi-square difference (linkage) was detected by substracting deviation due to individual gene segregation from the combined deviation due to joint segregation of genes.

RESULTS AND DISCUSSION

Results of the tests in seedlings and adult plants on parental lines and F_1s involving Sel.212 against pathotypes 77-1, 77-3, 77-4 and 77-5 are presented in

Table 1. Sel. 212 was resistant both at seedling and adult plant stage in contrast to Agra Local. However, Chinese Spring was susceptible at seedling but showed moderate resistance at adult plant stage. Due to this adult plant resistance of Chinese Spring crosses of Sel. 212 with Chinese Spring were tested only in seedlings. The F_1 seedlings of crosses Sel. 212 × AL, Sel. 212 × CS and CS × Sel. 212 were susceptible to all the four pathotypes indicating that resistance gene(s) in Sel. 212 is recessive. However, F_1 of cross Sel. 212 × AL showed moderate resistance (50 MR) in adult plant tests suggesting the operation of adult plant resistance in Sel. 212.

Results of the tests on the F_2, F_3, BC_1F_1 and BC_1F_2 obtained from the crosses involving Sel. 212, Agra Local and Chinese Spring against pathotype 77-1, 77-3, 77-4 and 77-5 of *P. recondita tritici* at seedling stage and with pathotype 77-5 at adult stage are presented in Table 2. When tested with pathotype 77-1, the observed F_2 segregation in two phenotypic classes of different crosses fitted well in an expected ratio of 1 Resistant (R) : 3 susceptible (S) (χ^2 = 0.003 - 1.186, P = 0.95 - 0.20) demonstrating that the resistance is controlled by single recessive gene. Analysis of pooled data of three crosses also segregated in the same ratio with non-significant heterogeneity among the crosses (χ^2 = 0.683 at P > 0.70). Data from the reciprocal crosses revealed the absence of cytoplasmic influence in controlling resistance. The F_3 families distribution (24R : 42Seg : 14S) conformed with 1R : 2Seg : 1S segregation pattern for single locus. The susceptibility of all 65 seedlings of the backcross (Sel.212 × AL) × AL was expected as the resistance was determined to be recessive. The F_1 seedling from the backcrosses (Sel. 212 × AL) × Sel. 212 and (Sel. 212 × CS) × Sel. 212 segregated into 1R : 1S ratio confirming that resistance is controlled by single recessive gene. The distribution of the BC_1F_2 families from the cross (Sel. 212 × AL) × AL into 1Seg : 1S ratio was non-significant (χ^2 = 0.571, P > 0.30) which further confirmed the presence of single recessive resistance gene.

The results with pathotypes 77-3, 77-4 and 77-5 also suggested inheritance of single recessive gene to each of the pathotypes. The conclusion that whether the same or different gene provide resistance to all the four pathotypes was provided by joint segregation tests. Correlated behaviour of the common F_3 and the BC_1F_2 families with pathotypes 77-1, 77-3, 77-4 and 77-5 was analysed and presented in Table 3. The distribution of the common set of F_3 families into resistant, segregating and susceptible classes against pathotype 77-1 was compared with distribution of the F_3 families tested with pathotypes 77-3, 77-4 and 77-5. Highly significant chi-square linkage values ($\chi^2_L = 142.0 - 152.775$, P < 0.001) indicated strong association of the resistance gene effective to 77-1 with resistance gene(s) effective to 77-3, 77-4 and 77-5. The significant chi-square values for linkage when same set of the BC_1F_2 families

Table 3. Correlated behaviour of F_3 and BC_1F_2 families from the cross of Sel. 212 with Agra Local against pathotypes 77-1, 77-3, 77-4 and 77-5

		Number of F_3 families				$\chi^2_{(1:2:1)}$	Number of BC_1F_2 families			$\chi^2_{(1:1)}$
		Res.	Segr.	Susc.	Total		Segr.	Susc.	Total	
Pathotype 77-1										
Pathotype 77-3	Res.	23	0	0	23					
	Segr.	1	42	1	44	3.3, 2 df, P > 0.10	14	0	14	0.000 1df, P > 0.99
	Susc.	0	0	13	13		2	12	14	
	Total	24	42	14	80		16	12	28	
$\chi^2_{1:2:1}$ = 2.7, 2 df, P > 0.20							$\chi^2_{1:1}$ = 0.571, 1 df, P > 0.03			
$\chi^2_{(Compound)1:2:1:2:4:2:1:2:1}$ = 148.0, 8 df, P < 0.001							$\chi^2_{(Compound)1:1:1:1}$ = 21.143, 3 df, P < 0.001			
$\chi^2_{difference(linkage)}$ = 142.0, 4 df, P < 0.001							$\chi^2_{difference\ (Linkage)}$ = 20.572, 1 df, P < 0.001			
Pathotype 77-4	Res.	24	1	0	25					
	Segr.	0	41	0	41	3.075, 2df, P > 0.20	13	0	13	0.143, 1 df, P > 0.70
	Susc.	0	0	14	14		3	12	15	
	Total	24	42	14	80		16	12	28	
$\chi^2_{1:2:1}$ = 2.7, 2 df, P<0.20							$\chi^2_{1:1}$ = 0.571, 1 df, P> 0.30			
$\chi^2_{(Compound)1:2:1:2:4:2:1:2:1}$ = 158.55, 8 df, P < 0.001							$\chi^2_{(Compound)1:1:1:1}$ = 18.0, 3 df, P < 0.001			
$\chi^2_{difference(Linkage)}$ = 152.775, 4 df, P < 0.001							$\chi^2_{difference(Linkage)}$ = 17.286, 1 df, P < 0.001			
Pathotype 77-5	Res.	23	0	0	23					
	Segr.	1	42	0	43	2.475, 2df, P > 0.20	14	0	14	0.000, 1 df, P > 0.99
	Susc.	0	0	14	14		2	12	14	
	Total	24	42	14	80		16	12	28	
$\chi^2_{1:2:1}$ = 2.7, 2 df ,P> 0.20							$\chi^2_{1:1}$ = 0.571, 1 df, P>0.30			
$\chi^2_{(Compound)1:2:1:2:4:2:1:2:1}$ = 153.3, 8df, P<0.001							$\chi^2_{1:1:1:1}$ = 21.143, 3 df, P<0.001			
$\chi^2_{difference(Linkage)}$ = 148.125, 4 df, P<0.001							$\chi^2_{difference(Linkage)}$ = 20.572, 1 df, P < 0.001			

(*Table 3 Contd.*)

		Number of F_3 families				$\chi^2_{(1:2:1)}$	Number of BC_1F_2 families			$\chi^2_{(1:1)}$
		Res.	Segr.	Susc.	Total		Segr.	Susc.	Total	
Pathotype 77-3										
Pathotype 77-4	Res.	23	2	0	25					
	Segr.	0	41	0	41	3.07, 2df, P>0.20	13	0	13	0.14, 1 df, P>0.70
	Susr.	0	1	13	14		1	14	15	
	Total	23	44	13	80		14	14	28	
$\chi^2_{1:2:1}$ = 3.3, 2 df, P>0.10							$\chi^2_{1:1}$ = 0.000, 1 df, P>0.99			
$\chi^2_{(Compound1:2:1:2:4:2:1:2:1}$ = 144.15, 8 df, P<0.001							$\chi^2_{(Compound1:1:1:1}$ = 24.28, 3 df, P<0.001			
$\chi^2_{difference(Linkage)}$ = 137.775, 4 df, P<0.001							$\chi^2_{difference(Linkage)}$ = 24.14, 1 df, P<0.001			
Pathotype 77-5	Res.	23	0	0	23					
	Segr.	0	43	0	43	2.47, 2df, P>0.20	14	0	14	0.00, 1 df, P>0.99
	Susr.	0	1	13	14		0	14	14	
	Total	23	44	13	80		14	14	28	
$\chi^2_{1:2:1}$ = 3.3, 2 df, P>0.10							$\chi^2_{1:1}$ = 0.000, 1 df, P>0.99			
$\chi^2_{(Compound)1:2:1:2:4:2:1:2:1}$ = 152.15, 8 df, P<0.001							$\chi^2_{(Compound)1:1:1:1}$ = 28.0, 3 df, P<0.001			
$\chi^2_{difference(Linkage)}$ = 146.375, 4 df, P<0.001							$\chi^2_{difference(Linkage)}$ = 28.0, 1 df, P<0.001			
Pathotype 77-4										
Pathotype 77-5	Res.	23	0	0	23					
	Segr.	2	41	0	43	2.47, 2df, P>0.20	13	1	14	0.00, 1 df, P>0.99
	Susr.	0	0	14	14		0	14	14	
	Total	25	41	14	80		13	15	28	
$\chi^2_{1:2:1}$ = 3.075, 2 df, P>0.10							$\chi^2_{1:1}$ = 0.14, 1 df, P>0.70			
$\chi^2_{(Compound)1:2:1:2:4:2:1:2:1}$ = 149.45, 8 df, P<0.001							$\chi^2_{(Compound1:1:1:1}$ = 24.28, 3 df, P<0.001			
$\chi^2_{difference(Linkage)}$ = 143.9, 4 df, P<0.001							$\chi^2_{difference(Linkage)}$ = 24.14, 1 df, P<0.001			

were tested with same set of pathotypes exhibited close association of gene for resistance to 77-1 with those effective to 77-3, 77-4 and 77-5. Similar close associations were observed when distribution of the F_3 and the BC_1F_2 families for pathotype 77-3 with pathotype 77-4 and 77-5 and pathotype 77-4 with pathotype 77-5 were compared.

The comparison of leaf rust reaction of F_3 and the BC_1F_2 families against pathotypes 77-1, 77-3, 77-4 and 77-5 suggested a strong association of the resistance genes effective to these four pathotypes. It is highly improbable that four different genes that are differentiated by these four pathotypes are present in Sel. 212 which would imply that each resistance gene is effective to one pathotype and ineffective to other three pathotypes. A logical conclusion can therefore be drawn that it is only one gene that is most likely effective to all the four pathotypes. The odd discrepant F_3 and BC_1F_2 families may have resulted from misclassification of mesothetic reactions coupled with changes in infection types caused by temperature fluctuations. The difficulties in classifying seedling reactions in certain situations were also reported by Knott and McIntosh [8], Singh and McIntosh [9, 10] etc.

The F_1, F_2, F_3, BC_1F_1 and BC_1F_2 generations from the cross of Sel.212 with Agra Local and the parental lines were evaluated for terminal disease severity at adult plant stage against pathotype 77-5 under field conditions (Table 2). The F_2 population from the above cross contained 246 resistant (TR-50MR) and 68 susceptible (70S-90S) plants which fits into a 13R : 3S ratio suggesting the expression of one dominant and one recessive gene for resistance. The distribution of F_3 families into the expected 7R : 8Seg : 1S. ratio (χ^2 = 0.738, P > 0.30) confirmed digenic control of resistance. Intra-family segregation of F_3 families into 1R:3S and 3R:1S/13R:3S ratio further supports the conclusion that Sel.212 carries one dominant and one recessive gene for resistance against pathotype 77-5. 29 F_1 plants of the backcross (Sel.212 × AL) × AL segregated into 1R : 1S ratio suggesting an operation of a dominant adult plant resistance gene in Sel. 212 because in seedling tests all the BC_1F_1 plants were susceptible. 28 BC_1F_2 families segregated for 3Seg : 1S ratio confirming the digenic control of resistance. This analysis revealed the presence of one recessive seedling resistance gene presumably derived from rye and providing resistance throughout the plant life. In addition Sel.212 carries one dominant adult plant resistance gene which is effective only at adult stage of plant growth. Chinese Spring one of the parent in the development of Sel. 212 carries *Lr12*, *Lr31* and an adult plant resistance gene *Lr34* [11]. *Lr12* and *Lr31* are, however, not effective to pathotype 77-5. It is, therefore, likely that the dominant adult plant resistance gene identified in the present study may be *Lr34* derived from Chinese Spring.

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