Genetics

Linkage Between Leaf Rust Resistance Genes and Morphological Markers in Barley

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ABSTRACT

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Studies were undertaken to map the chromosome location of leaf rust resistance genes *Rph*3, *Rph*7, and *Rph*12 in the barley cultivars Estate, Cebada Capa, and Triumph using various morphological markers. Crosses were made between genotypes resistant to *Puccinia hordei* and leaf rust susceptible morphological marker stocks. F_2 plants were evaluated for reaction (infection type) to *P. hordei*, and presence of morphological marker characters. Segregation data indicated the position of the *Rph*3 allele in Estate barley on the long arm of chromosome 1.

A linkage distance of 9.7 ± 4.2% was found between the *Rph*3 and X_a loci. The independence of segregation of *Rph*3 with *n* and *lk*2 suggests that the *Rph*3 locus is more distal than the X_a locus on the long arm of chromosome 1. Linkage between *Rph*7 in Cebada Capa and markers on chromosomes 1 to 7 was not observed. A single resistance gene was identified in Triumph and was designated *Rph*12. The *Rph*12 locus was linked with the *r* and *s* loci on chromosome 7, with recombination values of 26.1 ± 2.3% and 39.5 ± 2.9%, respectively.

Barley (*Hordeum vulgare* L.) is a diploid species ($2n = 2x = 14$) that is often used in genetic and cytogenetic studies. Genes conditioning resistance to *Puccinia hordei* G. Otth have been identified in barley from different sources since the early studies of Waterhouse (19) and Henderson (4). These genes were designated as *Rph* genes (formerly *Pa* genes) (2,11,12). Among the known resistance genes, *Rph*3 from Estate, *Rph*7 from Cebada Capa, and *Rph*12 from Triumph have been used in various barley breeding programs (1,8,14).

The use of morphological markers in mapping *Rph* genes has not been successful. *Rph*4 was mapped on chromosome 5 using the *Reg*1 (*Ml*-a) locus as a genetic marker (10). Tuleen and McDaniel (17) used six primary trisomics (all except chromosome 1) to associate *Rph*1 with chromosome 2 and *Rph*7 with chromosome 3. They also confirmed the association of *Rph*4 with chromosome 5 using the same method. Tan (15) used trisomics to confirm the associations of *Rph*4 and *Rph*7 on chromosomes 5 and 3, respectively. The *Rph*3 locus was not mapped on chromosomes 3, 4, 5, 6 or 7; thus, by inference, it would be on either chromosome 1 or 2. The objective of this study was to map the chromosome location of the leaf rust resistance genes *Rph*3, *Rph*7, and *Rph*12 using morphological markers distributed across the seven barley linkage groups.

MATERIALS AND METHODS

Plant materials. Barley genotypes Estate (CI 3410), Cebada Capa (CI 6193), and Triumph (PI 268180) were used as the donors of *Rph* genes in crosses. Morphological marker stocks (20) were used as susceptible parents (Table 1). Genotypes with *Rph* genes were crossed to lines having one to several morphological marker genes. Crosses were made in the greenhouse in 1987, 1988, and 1989, and F_1s were grown in the field. The parent with the X_a gene was heterozygous because the homozygous condition for this allele produces xantha lethal (plant dies at the second leaf stage). With the F_1 progeny, plants without the X_a allele (green plants) were eliminated, and the heterozygous chlorina plants were grown to produce F_2 seed. Seed of F_2 populations was sown in clay pots (15 cm diameter) filled with No. 1 Sunshine Mix (Fisons Horticulture, Vancouver, Canada). Three to five seeds were sown in each pot. Plants were grown in the greenhouse or growth chamber with a photoperiod of 12 h at 22 C. For most crosses, the morphological markers were scored when the phenotype for each particular genetic marker was best expressed.

Rust inoculation and evaluation. Culture ND8702 of *P. hordei* race 8 was used throughout the experiments. Preliminary evaluations of parental lines indicated that ND8702 was avirulent on Estate, Cebada Capa, and Triumph and virulent on the morphological marker stocks. Morphological marker genes had no obvious effects on leaf rust infection types. Parental, F_1, and F_2 plants were inoculated 7 days after planting when the primary leaf was fully expanded. A urediniospore suspension in Soltrol 170 oil (Phillips Petroleum, Borger, TX) was applied to plants at a rate of 3 mg of spores per 5 ml of oil per 100 plants.

The rating scale of Levine and Cherewick (9) was used to score infection types (ITs) of the parental, F_1, and F_2 plants 10 to 14 days after inoculation, depending upon rust development. Plants with ITs of 23− or higher were considered susceptible, and those with ITs of 0;, 0;1, 12, or 21 resistant. Reaction to

Puccinia graminis Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn. was evaluated using the TPM pathotype between the boot and heading stages of growth on an F_2 population that was tested for leaf rust reaction at the seedling stage.

Data analysis. Complete dominance of the dominant allele was assumed for most morphological marker loci and for *Rph* genes for the purpose of analysis, although incomplete dominance was common for the *Rph* genes under study (Y. Jin, *unpublished*). In F_2 populations, the expected ratio of phenotypes was 3:1 for dominance versus recessiveness for one pair of genes and 9:3:3:1 for two pairs of genes when independent segregation was expected, except for the X_a cross. The chi-square (χ^2) method was used to test the hypothesis of independent segregation in the F_2 populations, and the method of maximum likelihood was used to calculate linkage intensities from F_2 data. A program was written in SAS (Statistical Analysis System, SAS Institute) to facilitate computations of chi-square values, linkage intensities for repulsion and coupling crosses based on several methods, and standard errors. Homogeneity among different crosses of the same linkage relation was tested, and data were pooled when the test was not significant.

The methods of calculating recombination and chi-square values in the X_a cross were modified, since plants homozygous for X_a died as seedlings, and plants heterozygous for X_a produced additional classes in the F_2 population. The linkage was determined based on the total chi-square value of five-class segregation with four degrees of freedom rather than the chi-square value for linkage because of the incomplete data for resistant versus susceptible segregation. A maximum likelihood function was derived from a multinomial distribution function:

$$LP(a,b,c,d,e) = f(p,N,a,b,c,d,e)$$

where, p denotes the recombination fraction at the repulsion phase; a, b, c, d, and e denote the observed frequencies for F_2 phenotypes; and N denotes the size of the F_2 population. The five classes of phenotypes in the F_2 were resistant yellow (a), resistant green (b), susceptible yellow (c), susceptible green (d), and xantha seedlings (e). A quartic equation was derived based on the maximum likelihood function of the form:

$$2(a+b+c+d)p^4 - (a+2b+c+2d)p^3$$
$$-2(a-b)p^2 + (a+2c+2d)p - c - 2d = 0.$$

The population size (N) and the xantha type of plants (e) were eliminated in the process of differentiation of the maximum likelihood function for being constants. A program was written in FORTRAN 77 to solve the quartic equation for p numerically.

TABLE 1. Barley morphological marker genes, their characteristics, and associations with barley chromosomes used to determine linkage relationships for leaf rust resistance genes

Gene locus[a]	Phenotype or gene name	Chromosomal location[a]	Stock number or origin[b]
al	albino lemma (eburatum)	3 S	BGS 108
*br*1	brachytic dwarf	1 S	BGS 001
*cu*2	curly leaf	3 L	BGS 114
e	lemma-like glumes	2 S	BGS 057
f_c	chlorina seedling	1 S	BGS 002
*f*2	chlorina seedling	3 L	BGS 117
*f*3	chlorina seedling	5 S	BGS 220
*f*5	chlorina seedling	1 S	BGS 018
Cer-yy (*Gle*)	glossy spike	5 S	Cer-yy[849]
*gs*2 (*cer*-b)	glossy sheath/spike	3 L	BGS 352
*gs*3 (*cer*-a)	glossy sheath/spike	1 S	BGS 353
*gs*6 (*cer*-c)	glossy sheath/spike	4 S	BGS 356
li	ligule and auricle less	2 L	BGS 060
*lk*2	short awn	1 L	BGS 009
lnt	low number of tillers	3 L	BGS 118
*msg*2	male sterile	2 S	BGS 358
*msg*5	male sterile	3 S	BGS 361
*msg*10	male sterile	1 L	BGS 366
n	naked caryosis	1 L	BGS 007
*nec*1	necrotic spots	5 L	BGS 222
o	orange lemma base and nodes	6 L	BGS 254
r	semismooth awn	7 L	BGS 312
*Rpg*1	Resistance to stem rust	1 S	Bowman
s	short rachilla hairs	7 L	BGS 312
trd	third outer glume	5 L	BGS 202
uz	"uzu" (semibrachytic)	3 L	BGS 102
v	six-rowed spike	2 L	BGS 006
*wst*3	white stripe	3 L	BGS 103
wst,,k	white stripe	2 L	MR[c]
X_a	xantha seedling	1 L	OUM 215

[a]Gene loci and chromosomes are based on Sφgaard and Wettstein-Knowles (13).
[b]Most original stocks were backcrossed to Bowman two to four times.
[c]R. I. Wolfe's multiple recessive (MR) marker stock (20).

RESULTS

***Rph*3 linkage.** Of the 22 morphological markers used to test the linkage relationships with *Rph*3 on chromosome 1, only the X_a locus was linked with the *Rph*3 locus (Table 2). These data place *Rph*3 on the long arm of chromosome 1. The maximum likelihood estimate for the linkage distance was 9.7 ± 4.2% between the *Rph*3 and X_a loci. Since *Rph*3 was not linked with the *lk*2 and *n* loci on the long arm of chromosome 1, the *Rph*3 locus is probably located at a more distal position than the X_a locus.

***Rph*7 linkage.** No linkage was found between *Rph*7 and the morphological marker loci used in this study.

***Rph*12 linkage.** An incompletely dominant gene was detected in Triumph. This gene was designated *Rph*12. The *Rph*12 locus was found to be linked with the *r* and *s* loci on chromosome 7 with recombination values of 26.1 ± 2.3% and 39.5 ± 2.9%, respectively (Table 3). These data indicate that the *Rph*12 locus is located on the long arm of chromosome 7 beyond the *r* locus.

DISCUSSION

The linkage relationship between the *Rph*3 and X_a loci were indicated not only by a significant total chi-square value (χ^2_T, Table 2), but also by a significant chi-square value for segregation at the *Rph*3 locus (χ^2_{Rph3}). The lack of susceptible plants in the F_2 suggested that a major portion of xantha plants have the susceptible genotype (*rph3rph3*), although the exact χ^2_L cannot be calculated in this cross. The allele controlling the xantha trait (X_a) was first studied by Konishi (7), who detected linkage with the *lk*2 and *n* loci on chromosome 1. The X_a locus was placed on the most distal region on the long arm of chromosome 1 (16). Since *Rph*3 was not linked with *lk*2 or *n* (C1-3, 4), the likely position of this locus is distal to X_a. The position of the *Rph*3 locus can be better defined when other morphological markers on the distal half of long arm of chromosome 1 are identified.

TABLE 2. Segregation ratio of F_2 plants for *Rph*3 and X_a on chromosome 1

Phenotype	Frequency of phenotype: Observed	Expected	Ratio[a]
Resistant			
Green	156	101.4	3/16
Yellow	233	202.9	6/16
Susceptible			
Green	0	33.8	1/16
Yellow	20	67.6	2/16
Xantha seedling	132	135.2	4/16

[a]Calculated chi-square values were χ^2_{Rph3} 88.23 ($P < 0.01$) for segregation of *Rph*3 (based on a 3:1 ratio of resistant to susceptible viable plants), χ^2_{Xa} 4.17 for segregation of X_a (based on a 1:2:1 ratio of xantha to yellow to green plants), and χ^2_T 101.26 ($P < 0.01$) for cosegregation of *Rph*3 and X_a (based on a 3:6:1:2:4 ratio of resistant green to resistant yellow to susceptible green to susceptible yellow to xantha plants).

The significant χ^2_L between *Rph*3 and *wst*,,k (C2-5), and *lnt* (C3-4) were not conclusive for a linkage relationship, because the segregations in these two crosses did not follow a pattern of linkage when crosses were made in the coupling phase. The expression of *wst*,,k and *lnt* can be affected by environmental conditions. This may have contributed to the significant χ^2_L for these two crosses. The pooling of data resulted in the significant χ^2_L between *Rph*3 and *uz* (C3-5, 6), *Rph*3 and *gs*6 (C4-1, 2), and *Rph*3 and *o* (C6-1, 2), since tests of individual F_2 populations did not yield significant χ^2_L in these crosses. Misclassification might have occurred in some crosses involving *Rph*3 and *r*, as indicated by significant chi-square tests for segregation of single pairs of genes in some crosses (C2-1, 3, C7-1). The *Rph*3 gene is incompletely dominant (Y. Jin, *unpublished*), and the intermediate infection type can be affected by temperature. The semismooth awn character *r* is sometimes difficult to score, especially when plants are grown in the greenhouse. The significant χ^2 for *f*3 segregation may indicate that the *f*3 parent in this cross

TABLE 3. Segregation ratios of F_2 plants for *Rph* genes and for morphological markers from barley crosses

Cross	Parents[a]	Gene tested[b]		Phenotype[c] and observed frequency						
		A	B	AB	Ab	aB	ab	χ^2_A[d]	χ^2_B	χ^2_L
Chromosome 1										
C1-1	BGS001/Estate	*Rph*3	*br*1	335	98	93	41	0.56	0.07	3.36
C1-2	BGS353/Estate	*Rph*3	*gs*3	328	105	97	37	0.56	0.00	0.59
C1-3	BGS009/Estate	*Rph*3	*lk*2	318	87	99	30	0.20	2.72	0.19
C1-4	BGS007/Estate	*Rph*3	*n*	305	97	92	31	0.69	0.11	0.06
C1-5	Bowman/Estate	*Rph*3	*Rpg*1	196	81	71	17	0.15	0.67	3.49
C1-6	BGS001/Cebada Capa	*Rph*7	*br*1	314	100	91	42	0.14	0.27	2.87
C1-7	BGS002/Cebada Capa	*Rph*7	f_c	555	185	155	60	3.15	0.22	0.65
C1-8	BGS353/Cebada Capa	*Rph*7	*gs*3	295	101	92	38	0.06	0.57	0.71
C1-9	BGS009/Cebada Capa	*Rph*7	*lk*2	361	108	107	33	1.31	1.11	0.03
C1-10	BGS366/Cebada Capa	*Rph*7	*msg*10	297	99	99	31	0.02	0.02	0.07
C1-11	BGS007/Cebada Capa	*Rph*7	*n*	338	129	96	40	1.92	2.95	0.10
C1-12	Triumph/BGS009	*Rph*12	*lk*2	295	92	104	29	0.09	0.83	0.22
Chromosome 2										
C2-1	BGS057/Estate	*Rph*3	*e*	278	84	122	25	4.09*[e]	3.49	2.89
C2-2	BGS060/Estate	*Rph*3	*li*	297	94	116	28	1.05	1.38	1.36
C2-3	BGS358/Estate	*Rph*3	*msg*2	271	91	121	26	4.09*	1.10	3.75
C2-4	BGS006/Estate	*Rph*3	*v*	308	97	94	35	0.20	0.02	0.52
C2-5	MR/Estate	*Rph*3	*wst*,,k	215	81	69	13	2.20	0.01	4.09*
C2-6	BGS057/Cebada Capa	*Rph*7	*e*	400	121	124	42	0.26	0.59	0.30
C2-7	BGS358/Cebada Capa	*Rph*7	*msg*2	402	120	124	42	0.28	0.78	0.37
C2-8	BGS006/Cebada Capa	*Rph*7	*v*	399	121	122	44	0.24	0.33	0.71
C2-9	MR/Cebada Capa	*Rph*12	*wst*,,k	212	70	68	28	0.03	0.17	0.73
Chromosome 3										
C3-1,2,3[f]	BGS108/Estate	*Rph*3	*al*	741	260	273	81	0.92	0.02	1.36
C3-4	BGS118/Estate	*Rph*3	*lnt*	301	107	88	47	0.01	3.27	3.95*
C3-5,6[f]	BGS102/Estate	*Rph*3	*uz*	630	215	203	95	0.70	2.74	5.19*
C3-7	BGS108/Cebada Capa	*Rph*7	*al*	386	110	104	41	1.94	0.71	2.21
C3-8	Cebada Capa/BGS114	*Rph*7	*cu*2	336	108	119	29	0.00	1.09	1.32
C3-9	BGS117/Cebada Capa	*Rph*7	*f*2	298	110	103	48	1.21	3.18	1.65
C3-10	BGS352/Cebada Capa	*Rph*7	*gs*2	234	75	75	21	0.32	0.36	0.20
C3-11	BGS118/Cebada Capa	*Rph*7	*lnt*	171	73	108	64	59.28**	13.96**	11.12**
C3-12	BGS361/Cebada Capa	*Rph*7	*msg*5	329	107	113	50	1.56	0.47	2.63
C3-13,14[f]	BGS102/Cebada Capa	*Rph*7	*uz*	562	182	183	64	0.00	0.02	0.21
C3-15	BGS103/Cebada Capa	*Rph*7	*wst*3	196	49	51	23	0.55	1.00	3.70
C3-16	Triumph/BGS108	*Rph*12	*al*	302	92	105	30	0.08	1.06	0.08
Chromosome 4										
C4-1,2[f]	BGS356/Estate	*Rph*3	*gs*6	549	204	229	62	4.60*	0.13	3.92*
C4-3	BGS356/Cebada Capa	*Rph*7	*gs*6	395	129	123	43	0.33	0.01	0.11
Chromosome 5										
C5-1,2[f]	Cer-yy^{849}/Estate	*Rph*3	*Cer*-yy	403	134	131	43	0.11	0.00	0.00
C5-3	Estate/BGS220	*Rph*3	*f*3	270	33	82	7	1.10	45.77**	0.04
C5-4	Estate/BGS222	*Rph*3	*nec*1	313	99	106	29	0.03	0.75	0.34
C5-5	Estate/BGS202	*Rph*3	*trd*	283	99	95	25	0.32	0.02	1.21
Chromosome 6										
C6-1,2[f]	BGS254/Estate	*Rph*3	*o*	611	162	196	74	0.44	3.13	4.39*
C6-3,4,5[f]	BGS254/Cebada Capa	*Rph*7	*o*	1063	322	340	92	1.45	4.76*	0.55
C6-6	Triumph/BGS254	*Rph*12	*o*	290	97	110	23	0.09	1.03	3.29
Chromosome 7										
C7-1	BGS312/Estate	*Rph*3	*r*	293	117	81	43	0.90	7.01**	1.54
C7-2	BGS312/Estate	*Rph*3	*s*	310	100	95	29	0.90	0.20	0.04
C7-3	BGS312/Cebada Capa	*Rph*7	*r*	286	97	98	47	1.71	1.45	3.24
C7-4,5,6,7[f]	BGS312/Cebada Capa	*Rph*7	*s*	1378	416	422	141	1.56	2.35	0.83
C7-8	Triumph/BGS312	*Rph*12	*r*	325	62	58	75	0.09	0.50	87.52**
C7-9	Triumph/BGS312	*Rph*12	*s*	296	91	80	53	0.09	2.01	14.44*

[a]The parents given were the original sources for *Rph* and marker genes. Morphological stocks were backcrossed to Bowman two to four times.
[b]Genes tested were A, the *Rph* gene, and B, a morphological marker.
[c]Phenotypes represent dominant alleles A and B, dominant allele A and recessive allele b, recessive allele a and dominant allele B, and a double recessive genotype.
[d]χ^2_A, χ^2_B, and χ^2_L represent the calculated chi-square values for genes a, b, and linkage, respectively. $\chi^2_{0.05}(1) = 3.84$ and $\chi^2_{0.01}(1) = 6.63$.
[e]* And ** represent significance at $P = 0.05$ and $P = 0.01$, respectively, for a χ^2.
[f]Data were from more than one cross pooled after homogeneity test.

(C5-3) was heterozygous.

The independence of segregation between *Rph*7 and most morphological marker loci on chromosome 3 is especially puzzling, because trisomic studies confirmed the association of *Rph*7 with chromosome 3 (15,17). The markers studied on chromosome 3 are well distributed throughout the chromosome region. Linkage between *Rph*7 and *lnt* indicated in the cross C3-11 is very questionable because of the abnormal segregation for both loci. The excessive number of susceptible plants in the F_2 suggested that the resistant parent might be heterozygous. The significant χ^2 for *lnt* segregation may be due to environmental conditions, as discussed previously.

The two or more gene model for reaction to *P. hordei* in Triumph (18) could not be confirmed in this study, because one incompletely dominant gene was detected in the crosses (C7-8 and C7-9) involving Triumph using race 8 of *P. hordei*. It is likely that one of the reported genes in Triumph is ineffective against this race. Data from allelism tests (18; Y. Jin, *unpublished*) indicate that the gene in Triumph is different from *Rph*3 and *Rph*9. Differential reactions of Triumph to various races of *P. hordei* (5,6; Y. Jin and B. J. Steffenson, *unpublished*) suggest a probable genotype that is different from other known *Rph* genes. On the basis of these observations and the unique linkage location found in the present study, this gene is not allelic to the genes *Rph*1 to *Rph*9. The gene in Triumph was designated *Rph*12, because *Rph*10 and *Rph*11 were assigned to leaf rust resistance genes from *H. spontaneum* by Feuerstein et al (2).

The mapping of genes to a unique location on specific barley chromosomes, as was done for *Rph*3, *Rph*4, *Rph*10, *Rph*11, and *Rph*12 in this and other studies, can eliminate the need for allelism tests that are required for designating new loci. Additionally, closely linked morphological markers may facilitate the selection of economically important traits in barley breeding, as suggested by Franckowiak (3), although the linkage relationships obtained from this study may not serve this purpose. Information concerning the linkage relationships of *Rph* genes in barley will enable breeders to efficiently transfer leaf rust resistance in their germ plasm.

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