LOCATION OF er PROVING ELUSIVE

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In 1971 I presented results suggesting that *ex,* conferring resistance to powdery mildew, is situated on chromosome 3 (3). There were indications of linkage between er and several genes presumed or known to reside on chromosome 3. The evidence for linkage with Gty was particularly convincing and was supported further by results from F3 progeny tests (4). Because chromosome 3 is a well marked chromosome with many good seedling markers, it would seem a routine matter to fix the position of er with some precision. With that in mind, I constructed populations segregating for er and for markers along much of the known length of chromosome 3, from chl-6 on one end to tac on the other. Markers used were: tac apu st ***b*** bulf chi-6. Segregating populations were grown under controlled conditions in a growth chamber. Heavily infected susceptible plants were introduced into the chamber when the segregating F2 populations were in the early seedling stage, the fungal spores being spread by air currents generated by fans in the air handling system. Susceptible and resistant check populations were distributed throughout the chamber. Disease development was strong and uniform on susceptible checks and segregants.

The anticipated close linkage between er and one or more of the markers used did not materialize. Only a slight indication of linkage between b and er was found in one population (Table 1). A similar experiment was conducted in 1970 and it, too, failed to demonstrate linkage among er, st, and b (Table 2b, c). If er and Gty reside in chromosome 3 then presumably they lie distal to b, near gl and rag. However, chi-6 is supposed to be distal to b, yet there was no evidence of linkage between er and chi-6. This, therefore, calls into question my earlier findings (3) and further experiments are required to settle the issue. Possible involvement of a chromosomal interchange in the experiment reported in 1971 (3) cannot be excluded.

Our source of er traces to the variety 'Stratagem'. Harland (I) found a source of powdery mildew resistance in a remote site in the Andes mountains of Peru. Resistance was shown to be monogenic recessive with evidence of linkage with A on chromosome 1, the recombination fraction in and F3 being 35%. My calculations of the linkage chi-squares for his data are 4.80 and 4.42 for the F2 and F3 populations, respectively, both being significant at 0.05 but not at 0.01. His data also demonstrated the known and accepted linkage between A and Lf so his populations constituted a three point test. Because there was no evidence of linkage between Lf and er, he placed Lf distal to A (viz. Lf -12 -A-35-Er).

Recently I have acquired P.I. 185183 from the Northeast Regional Plant Introduction Station in Geneva, NY. The accession showed resistance to powdery mildew in my tests. Available data indicate that S. C. Harland presented the accession to the USDA in November 1949. Thus it may be the line representing the source of resistance referred to in liar land's 1948 paper but proof for that supposition is lacking. The F1 , F2, and subsequent progenies from the cross between P.I. 185183 and one of my er lines were all resistant. It would appear therefore that P.I. 185 183 carries the same gene for resistance as that carried by Stratagem. My resistant parental line carried st and bulf among other markers and these of course segregated In the F of the cross and the data are included to show normal, expected behavior (Table 3).

Since the er gene in the Geneva material possibly, even likely, is the same as that discovered by Harland, the same linkage relations should apply

to both sources. Since Linkage of er on chromosome 3 is still problematical, attention quite rightly should again focus on Harland's claim of linkage between er and A on chromosome 1. However, I have not been able to corroborate his findings either. For example, the same 42 F3 progenies from the testcross (A Gty Er x a gty er) x a gty er that demonstrated a close linkage between Gty and Er (4) showed independence between A and Er, the distribution of F3 families being as follows: A Er 14 : A er 11 : a Er 9 : a er 8. Moreover, results of the 1970 experiment (Table 2a) give no indication of linkage between A and Er.

The evidence for a second major gene, er-2, controlling resistance to powdery mildew (2) is subject to debate. Heringa et al. considered SVP 951 and SVP 952, both from Peru, as resistant but the lines showed heavy stem attack, so presumably the action of er-2 is individually identifiable on the basis of the differential disease reaction on leaves vis-a-vis stems. Yet Table 7 in their paper, showing the results of the cross between SVP 942 (Geneva line) and SVP 952, lists the classes as very strong, strong, weak and healthy, i.e. the plants were not placed in categories based on stem vs leaf reaction. Solely from the evidence presented in their paper, therefore, it may be just as reasonable to conclude that SVP 951 and SVP 952 carry polygenes for resistance as described by Hammerlund. That differences in reaction to powdery mildew exist among susceptible (i.e. Er) lines is well known to many breeders and others. Combinations of polygenes with er may therefore account for some of the observed differences in degree of disease reaction noted by Heringa et al. In my experience with the er gene derived from Stratagem, disease reaction ranges from complete absence of disease symptoms to the presence of fungal colonies over much of the plant, depending on the prevailing environmental conditions. Nevertheless, whenever susceptible and resistant plants are exposed to the same conditions, the resistant (i.e. er) plants are qualitatively distinguishable from Er plants.

Table 1. Joint segregation analysis of the cross St Chi-6 B Er x st chi-6 b er.

St	Chi-6	B	Er		Gene pair	Chi-square X	Chi-square Y	Linkage	Recomb. fract.
+	+	+	+	121					
+	+	+	-	32					
+	+	-	+	5	St Chi-6	1.14	0.11	29.95**	26.9
+	+	-	-	3	St B	1.14	0.60	29.03**	26.9
+	-	+	+	7	St Er	1.14	0.03	1.69ns	-
+	-	+	-	3	Chi-6 B	0.11	0.60	94.72**	13.9
+	-	-	+	15	Chi-6 Er	0.11	0.03	1.47ns	-
+	-	-	-	5	B Er	0.60	0.03	6.21*	38.6
-	+	+	+	15					
-	+	+	-	4					
-	+	-	+	3					
-	+	-	-	3					
-	-	+	+	7					
-	-	+	-	0					
-	-	-	+	12					
-	-	-	-	10					
				245					

(Population: A285-360)

*Significant at 0.05; ** significant at 0.01.

Table 2. Analysis of populations from crosses: a er St _B x A Er St B.

Data are from an experiment conducted in 1970, heretofore unreported, in which Er was tested for linkage with A on chromosome 1 (a), and with St and B on chromosome 3.

(a)

Gene pair	XY	Xy	xY	xy	N	Chi-square X	Chi-square Y	Linkage	Recomb. fract.	S.E.
A Er	318	116	85	38	557	2.53	2.08	0.65ns	-	-

(Populations: C270-240-258)

(b)

St	Er	B	No.
+	+	+	147
+	+	-	31
+	-	+	47
+	-	-	11
-	+	+	19
-	+	-	18
-	-	+	11
-	-	-	8
			292

Gene pair	Chi-square X	Chi-square Y	Linkage	Recomb. fract.	S.E.
St Er	5.28	0.29	2.04ns	-	-
St B	5.28	0.46	20.77**	31.8	3.4
Er B	0.29	0.46	0.10ns	-	-

(Populations: C270-244-258)

(c)

Gene pair	XY	Xy	xY	xy	N	Chi-square X	Chi-square Y	Linkage	Recomb. fract.	S.E.
St Er	332	107	85	39	563	2.66	0.26	2.26ns	-	-

(Populations: C270-244-258)

Table 3. Joint segregation in F_2 for genes St and Bulf.

Gene pair	XY	Xy	xY	xy	N	Chi-square X	Chi-square Y	Linkage	Recomb. fract.	S.E.
St Bulf	115	18	20	20	173	0.32	0.85	21.04**	26.7	4.0

(Population: B285-513-519)

1. Harland, S. C. 1948. Heredity 2:263-269.
2. Heringa, R. J., A. Van Norel, and M. F. Tazelaar. 1969. Euphytlca 18:163-169.
3. Marx, G. A. 1971. PNL 3:18-19.
4. Marx, G. A. 1974. PNL 6:30-31.