Aust. J. Biol. Sci., 1975, **28**, 531–8

Cytogenetical Studies in Wheat VIII.* Telocentric Mapping and Linkage Studies Involving *Sr22* and Other Genes in Chromosome 7AL

T. T. The and R. A. McIntosh

Plant Breeding Institute, University of Sydney, Sydney, N.S.W. 2006.

Abstract

The gene *Sr22* conditioning low reaction to *Puccinia graminis tritici* was located by telocentric mapping on chromosome 7AL at 27 ± 4 crossover units from the centromere. *Sr22* was approximately 2 crossover units from *cn-A1* (*chlorina*) and 41 units from *Pm1* for low reaction to *Erysiphe graminis tritici*; however, the order of *Sr22* and *cn-A1* relative to *Pm1* was not resolved. Maximum recombination between *Pm1*, *Lr20* and *Sr15*, for low reactions to *E. graminis tritici*, *P. recondita* and *P. graminis tritici* respectively, was estimated to be 0·3 crossover units at the 95% probability level.

Abnormal segregation at the *Sr22* and *cn-A1* loci was attributed to differential gametic transmission of *Sr22* but this had little effect on linkage estimates.

Introduction

The (1973) and Kerber and Dyck (1973) described *Sr22*, which confers resistance to certain cultures of *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn. in wheat (*Triticum aestivum* L.), and located it on chromosome 7A. The long arm of this chromosome also bears *cn-A1* determining *chlorina* phenotype (Sears and Sears 1968; Pettigrew and Driscoll 1970) and *Pm1* which confers resistance to certain cultures of *Erysiphe graminis* D.C. f. sp. *tritici* emend. Marchal (Sears and Briggle 1969). *Pm1* is closely linked with *Lr20*, which confers resistance to certain cultures of *P. recondita* Rob. ex Desm. (Waterhouse 1930; Watson and Baker 1943; Pugsley and Carter 1953), and with *Sr15* (Watson and Luig 1966). The present studies concern the genetic relationships of *Sr22* with the chromosome 7A centromere and with genes located in this chromosome.

Materials and Methods

The parentages of the homozygous genetic stocks used in the linkage studies were as follows (W numbers refer to the Sydney University Wheat Accession Register):

(i) W3534, Marquis*5//Stewart*3/*T. monococcum* L. (Kerber and Dyck 1973), and W3589, Steinwedel W199*2//Spelmar*2/*T. monococcum* (The 1973), both with *Sr22 Cn-A1 pm1 lr20 sr15* having low reaction to *P. graminis* and normal leaf colour.

(ii) Kenya W744/*Chlorina*-1 F_4 selection with *sr22 cn-A1 Pm1 Lr20 Sr15* having low reaction to *E. graminis*, *P. recondita* and *P. graminis* and *chlorina* leaf colour.

(iii) Chinese Spring Monotelosomic 7AL (CSMT 7AL) (41t, i.e. 41 chromosomes including a telocentric) with *sr22 Cn-A1 pm1 lr20 sr15*.

* Part VII, *Aust. J. Biol. Sci.*, 1975, **28**, 201–11.

The following cultures of the pathogens were chosen to identify the corresponding alleles in segregating lines:

(i) *P. graminis* culture 62020 (strain 17-2,3,7) and culture 61352 (strain 116-2,3,7). For strain designation see Watson and Luig (1963, 1966). Both cultures are avirulent (infection type "2−") on seedlings with *Sr22* but virulent on seedlings with *Sr15* (infection type "4").

(ii) *P. graminis* culture 691042 (strain 194-1,2,3,5,6) which is avirulent on seedlings with *Sr22* and, at temperatures below 20°C, those with *Sr15* (infection type "X−").

(iii) *E. graminis* culture S.U.2 which is avirulent on genotypes with *Pm1* (McIntosh and Baker 1966).

(iv) *P. recondita* culture BCL29, strain 76-0 (Watson and Luig 1961), which is avirulent on seedlings with *Lr20* (infection type ";N").

The initial cross for cytogenetic analysis was CSMT 7AL/W3534. F_2 analysis of monosomic plants ($20''+1' = 41$, i.e. 20 bivalents plus 1 univalent) permitted the location of *Sr22* on chromosome 7A (The 1973). In order to confirm this and to determine the genetic distance of *Sr22* from the centromere, further analyses were conducted on F_2 populations derived from monotelodisomic ($20''+t1'' = 42t$) siblings of the same cross. Seedlings were scored for somatic chromosome constitutions by root tip analysis and tested for the presence or absence of *Sr22* using *P. graminis* culture 62020.

Linkage relationships of the various genes were based on two crosses involving the *cn-A1–Pm1* stock with W3534 and W3589. F_2 seedlings were classified for leaf colour at the first leaf stage and appropriately tagged. These were then inoculated with *P. graminis* culture 62020 and approximately 6 days later with *E. graminis* to enable phenotypic classification for *Sr22* and *Pm1* respectively. Classified seedlings were then separated into phenotypic classes and grown to maturity. F_3 populations were scored to verify the F_2 classifications and also to study the segregations of *Lr20* and *Sr15*. After classification for seedling colour as described above, separate sowings of each F_3 line were treated in three ways:

(i) Inoculation with *P. graminis* culture 62020 and *E. graminis* as previously carried out for the F_2 population.

(ii) Inoculation with *P. recondita* and *E. graminis* enabling classification for *Lr20* and *Pm1*.

(iii) Inoculation with *P. graminis* culture 691042 and *E. graminis* enabling classification for *Sr15*, *Sr22* and *Pm1*. In addition to the characteristic infection types produced on seedlings where *Sr15* and *Sr22* occurred singly, seedlings with both *Sr15* and *Sr22* produced a lower infection type (";1−") when inoculated with culture 691042 and held at 18–20°C for 7 days.

Recombination values were estimated by the Method of Maximum Likelihood (Allard 1956; Mather 1963).

Results

Telocentric Mapping of Sr22

Chromosome counts and seedling reactions for progenies of two monotelodisomic (42t) F_1 plants of cross CSMT 7AL/W3534 are presented in Table 1. In using F_2 populations for telocentric mapping three parameters have to be estimated: the male and female gametic transmission rates of the telocentric (p and q respectively) and the recombination frequency (r) between the gene and the centromere. The male and female gametic transmission rates for the corresponding normal chromosome will be $(1-p)$ and $(1-q)$ respectively. The F_2 zygotic frequencies from a monotelodisomic (42t) F_1 plant will be:

$$2n = 42 \qquad (1-p)(1-q)$$
$$2n = 42t \qquad p(1-q)+q(1-p)$$
$$2n = 42tt \qquad pq$$

If the transmission through the female is unbiased as expected, i.e. $q = \frac{1}{2}$, then the corresponding frequencies will be:

$$2n = 42 \qquad \tfrac{1}{2}(1-p)$$
$$2n = 42t \qquad \tfrac{1}{2}p+\tfrac{1}{2}(1-p) = \tfrac{1}{2}$$
$$2n = 42tt \qquad \tfrac{1}{2}p$$

hence the ratio $(2n = 42 + 2n = 42tt) : (2n = 42t) = 1 : 1$ and the ratio $(2n = 42) : (2n = 42tt) = (1-p) : p$. This latter ratio will be 1 : 1 only if normal and deficient male gametes are equally transmitted, i.e. $p = \frac{1}{2}$.

Table 1. Pooled chromosome counts and *Sr22* phenotypes in progenies from two monotelodisomic F_1 plants of the cross CSMT 7AL/W3534

Chromosome number[A]	Phenotype	Expected proportion[B]	Observed number
42	*Sr22*	$\frac{1}{2}(1-p)(1-r^2)$	59
42	*sr22*	$\frac{1}{2}(1-p)r^2$	7
42t	*Sr22*	$\frac{1}{2}(1-r+r^2)$	68
42t	*sr22*	$\frac{1}{2}(r-r^2)$	20
42tt	*Sr22*	$\frac{1}{2}pr(2-r)$	6
42tt	*sr22*	$\frac{1}{2}p(1-2r+r^2)$	15
	Total	1	175
Excluded			
41	*Sr22*	—	2
41t	*Sr22*	—	1
43tt	*Sr22*	—	2
	Total	—	5

[A] The letter 't' indicates inclusion of one telocentric, 'tt' indicates inclusion of two telocentric chromosomes.

[B] p = male transmission rate of telocentric; r = recombination value between *Sr22* and the centromere.

The observed ratio of $(2n = 42 + 2n = 42tt) : (2n = 42t)$ was 87 : 88, indicating normal female transmission rates. On this basis the expected F_2 distribution in terms of p and r can be tabulated (Table 1). Excluding five resistant F_2 plants which possessed unexpected numbers of chromosomes, the male gametic transmission rate for the telocentric can be estimated as

$$p = 21/(66+21) = 0{\cdot}24,$$

and its standard error is

$$s_{(p)} = [2p(1-p)/n]^{\frac{1}{2}} = 0{\cdot}05.$$

Using the phenotypic frequencies from Table 1 the following maximum likelihood equation for r is obtained:

$$59\left(\frac{-2r}{1-r^2}\right)+7\left(\frac{2}{r}\right)-68\left(\frac{1-2r}{1-r+r^2}\right)+20\left(\frac{1-2r}{r(1-r)}\right)+6\left(\frac{2(1-r)}{r(2-r)}\right)-15\left(\frac{2}{1-r}\right) = 0,$$

and from this, $r = 27\%$. Using the value $p = 0{\cdot}24$, a standard error for r was computed at 4%.

Linkage Relationships for Sr22, cn-A1 *and* Pm1

Although two crosses were studied only pooled data are presented (Tables 2 and 3) since the data were homogeneous with respect to the characters being studied.

In scoring for *Sr22*, *P. graminis* culture 61352 was used to test F_2 populations whereas culture 62020 was used on F_3 progenies; both are virulent on seedlings with

Table 2. Pooled F_2 phenotypic frequencies from two crosses involving a *cn-A1–Pm1* stock with W3534 and W3589

Phenotype	Frequency	Phenotypic ratio	χ^2 3 : 1[A]
Sr22 Cn-A1 Pm1	285		
Sr22 Cn-A1 pm1	113		
Sr22 cn-A1 Pm1	4		
Sr22 cn-A1 pm1	2		
sr22 Cn-A1 Pm1	4		
sr22 Cn-A1 pm1	3		
sr22 cn-A1 Pm1	174		
sr22 cn-A1 pm1	33		
Total	618		
		Sr22 : *sr22* = 404 : 214	30·55
		Cn-A1 : *cn-A1* = 405 : 213	29·53
		Pm1 : *pm1* = 467 : 151	0·11

[A] Value for significance at $P = 0{\cdot}01$ is 6·63.

Sr15. F_2 segregations for *Sr22* and *cn-A1* deviated significantly ($P < 0{\cdot}01$) from those expected on the basis of allelic differences at single loci. In the case of *Sr22* there was an excess of seedlings with the recessive phenotype. Similar behaviour is also shown for *Cn-A1* and *cn-A1*, but segregation for *Pm1* and *pm1* was normal.

Of 618 transplanted F_2 plants, 474 (approximately 75%) produced sufficient seed for progeny testing (Table 3). Complete correlation between genotypic classifications

Table 3. Pooled F_3 classification for two crosses involving a *cn-A1–Pm1* stock with W3534 and W3589

Sr22 Sr22 Genotype	Frequency	*Sr22 sr22* Genotype	Frequency	*sr22 sr22* Genotype	Frequency
Cn-A1 Cn-A1 Pm1 Pm1	13	*Cn-A1 Cn-A1 Pm1 Pm1*	0	*Cn-A1 Cn-A1 Pm1 Pm1*	0
Cn-A1 Cn-A1 Pm1 pm1	45	*Cn-A1 Cn-A1 Pm1 pm1*	2	*Cn-A1 Cn-A1 Pm1 pm1*	0
Cn-A1 Cn-A1 pm1 pm1	37	*Cn-A1 Cn-A1 pm1 pm1*	4	*Cn-A1 Cn-A1 pm1 pm1*	1
Cn-A1 cn-A1 Pm1 Pm1	0	*Cn-A1 cn-A1 Pm1 Pm1*	60	*Cn-A1 cn-A1 Pm1 Pm1*	1
Cn-A1 cn-A1 Pm1 pm1	2	*Cn-A1 cn-A1 Pm1 pm1*	116	*Cn-A1 cn-A1 Pm1 pm1*	0
Cn-A1 cn-A1 pm1 pm1	2	*Cn-A1 cn-A1 pm1 pm1*	55	*Cn-A1 cn-A1 pm1 pm1*	1
cn-A1 cn-A1 Pm1 Pm1	0	*cn-A1 cn-A1 Pm1 Pm1*	0	*cn-A1 cn-A1 Pm1 Pm1*	51
cn-A1 cn-A1 Pm1 pm1	0	*cn-A1 cn-A1 Pm1 pm1*	2	*cn-A1 cn-A1 Pm1 pm1*	63
cn-A1 cn-A1 pm1 pm1	0	*cn-A1 cn-A1 pm1 pm1*	3	*cn-A1 cn-A1 pm1 pm1*	16

Genotypic ratios

Sr22 Sr22 : *Sr22 sr22* : *sr22 sr22* = 99 : 242 : 133

Cn-A1 Cn-A1 : *Cn-A1 cn-A1* : *cn-A1 cn-A1* = 102 : 237 : 135

Pm1 Pm1 : *Pm1 pm1* : *pm1 pm1* = 125 : 230 : 119

based on progeny tests in F_3 and the F_2 phenotypes confirmed the validity of the F_2 results. However, the genotypic distributions for the genes *Sr22* and *cn-A1* based on progeny tests on the 474 surviving F_2 plants were not consistent with the F_2 distributions. Comparisons of survival frequencies in classified F_2 classes indicated that mortalities were not evenly distributed. Losses were greater in the *sr22* or *cn-A1* groups or both, reflecting the reduced fitness and later maturity of the chlorophyll-deficient *cn-A1* phenotypes as well as the field susceptibility of *sr22* phenotypes to the

P. graminis strains present in the nursery. Mortalities were unbiased within *Pm1* and *pm1* phenotypes.

F_2 and F_3 results consistently indicated linkage between the three loci. The abnormal ratios obtained for *Sr22* and *cn-A1* are attributed to the *Sr22* locus or genes linked in coupling with it. Close linkage with *Sr22* could account for the abnormal segregation of *cn-A1*. Kerber and Dyck (1973) also obtained abnormal segregation ratios involving *Sr22*. On the other hand, the total segregation ratio of 451 *Cn-A1* : 105 *cn-A1* for the 20 non-critical monosomic populations studied by Sears and Sears (1968), although significantly deviating from a 3 : 1 ratio ($P < 0{\cdot}01$), did not suggest an excess of *cn-A1* segregates. In addition, the F_2 segregation ratio obtained by McIntosh (unpublished data) in the course of production of the *cn-A1 Pm1* parent used in this study was 148 *Cn-A1* : 49 *cn-A1*, suggesting normal segregation (3 : 1) at a single locus.

A major consideration is the likely effect of abnormal gametic transmission rates on the estimation of recombination values. If differential transmission is involved then the transmission of gametes with different genotypes will be affected as to whether they possess *Sr22* or *sr22*. Assuming a normal female transmission rate of 0·5 and letting the male transmission rate of *sr22* be k, then from Table 2

$$0{\cdot}5k = \frac{\text{frequency of } sr22\ sr22}{\text{total frequency}} = \frac{214}{618},$$

$$k = 0{\cdot}7.$$

Hence the male transmission rate of *Sr22*, $(1-k)$, is 0·3.

Estimates of recombination values based on the F_3 data were calculated from equations formulated by Allard (1956), and Appendix 1 shows the equations used in the estimations from the F_2 data. The following tabulation shows the estimated recombination values (%) ± S.E.:

	F_2 adjusted for $k = 0{\cdot}7$	F_2, no adjustment	F_3, no adjustment
Sr22–cn-A1	1·8±0·5	1·9±0·6	2·0±0·5
Sr22–Pm1	40·6±3·1	40·7±3·3	38·2±2·1
cn-A1–Pm1	40·1±3·1	40·2±3·3	37·8±2·1

Although the adjustment to F_2 data was not necessarily completely valid, linkage estimates based on it indicated that abnormal segregation ratios had little effect on such estimates. The adjusted estimates were only slightly less than those made without adjustment for differential gametic transmission. The slight difference in estimates based on F_2 and F_3 data can be partly attributed to the differential survival of the various F_2 genotypes, especially those in groups with low frequencies. Due to the very close linkage of *Sr22* and *cn-A1* their order relative to *Pm1* cannot be resolved.

Association of Pm1, Lr20 *and* Sr15

Among the 474 F_3 lines no recombinants with respect to these loci were detected. Assuming that each line represents two fully analysed gametes, then no crossovers occurred in $2 \times 474 = 948$ gametes. At the 0·05 level of probability the maximum distance (r) between any two of these loci is given by

$$(1-r)^{948} = 0{\cdot}05,$$

$$r = 0{\cdot}0032 \text{ or } 0{\cdot}3 \text{ crossover units.}$$

Discussion

Sr22 was located in the long arm of chromosome 7A and gave a recombination value of $27 \pm 4\%$ with the centromere. *Sr22* and *cn-A1* recombined with an approximate frequency of 2% but their order relative to the centromere and *Pm1* was not resolved. *Pm1* showed 41% recombination with *Sr22* indicating that *Pm1* is 27+41 or approximately 68 crossover units from the centromere.

Driscoll (1966) discussed the F_2 method of telocentric mapping. Provided populations are completely scored for chromosome constitution, reliable estimates can be derived for the male and female transmission rates of the telocentric chromosome. For genes close to the centromere, the F_2 method is equally efficient as the backcross method but this relative efficiency decreases to 0·5 for genes independent of the centromere (Mather 1943). For *Sr22*, which is 27 units from the centromere, the relative efficiency is 0·65. However, the greatest advantage of the F_2 method is its practical usefulness following monosomic analysis. In monosomic analysis programs aimed at locating genes, monotelosomic parents are preferable to monosomics since univalent shift (Person 1956) can be more easily detected. If both monosomic and monotelodisomic siblings are grown from a cross, the progenies from monosomics can be used for monosomic analysis. When the particular chromosome is identified among the monosomic populations, the progenies from the desired monotelodisomics are already available and can be used for confirmation of the location and, if recombinants occur (i.e. if the gene occurs in the paired arm), for linkage analysis. If no recombinants are recovered there is verification of the location, but further crossing with the opposite telocentric member is required for mapping. These methods were used in this study and are used routinely in this laboratory.

The observation of Kerber and Dyck (1973) that *Sr22* was not inherited in a simple manner was confirmed in these studies. However, the differential gametic transmission had little effect on linkage estimates. No attempts were made to correct for differential transmission in the telocentric mapping cross since it was unknown if the effects on telocentric chromosomes were identical with those on entire chromosomes. Furthermore, differential gametic transmission had little effect on conventional linkage estimates and since its validity was not firmly established there appears to be no significant advantage in attempting to correct for it in telocentric mapping.

All Australian and North American cultures of *P. graminis tritici* appear to be avirulent on seedlings with *Sr22*. However, limited data from Israel and Eastern Europe suggest that rare variants with virulence occur in those regions. Although *Sr22* is highly recommended for use in breeding programs, breeders should strive to combine it with other resistance genes to give added protection against any rare mutants with virulence on *Sr22*. An understanding of the types of gene interaction between *Sr22* and other genes (such as *Sr15*) could assist in the selection of segregants combining *Sr22* with such genes.

The complete coupling linkage of *Pm1*, *Lr20* and *Sr15* confirms previous reports of very tight linkage (Watson and Baker 1943; Watson and Luig 1966). The maximum genetic distance of 0·3% separating any of these genes estimated in this study is lower than that which may be deduced from the results of Watson and Luig (1966) who reported two recombinants from 116 F_2 progenies, but the alleged recombinants from their study were never isolated and confirmed because the major interest at that time was the understanding and use of *Sr15*, rather than the possibility that gene(s) *Pm1*,

Lr20 and *Sr15* may be the same. If the latter situation is correct then the same gene product, or components of a single gene product, should be involved in specific host–pathogen interactions for these diseases.

Acknowledgments

Thanks are due to Dr E. P. Baker for his interest and encouragement in this study. Financial support was provided by the Rural Credits Development Fund of the Reserve Bank of Australia and the Wheat Industry Research Council.

References

Allard, R. W. (1956). Formulas and tables to facilitate the calculation of recombination values in heredity. *Hilgardia* **24**, 235–78.

Driscoll, C. J. (1966). Gene–centromere distances in wheat by aneuploid F_2 observations. *Genetics* **54**, 131–5.

Kerber, E. R., and Dyck, P. L. (1973). Inheritance of stem rust resistance transferred from diploid wheat (*Triticum monococcum*) to tetraploid and hexaploid wheat and chromosome location of the gene involved. *Can. J. Genet. Cytol.* **15**, 397–409.

Mather, K. (1943). 'Statistical Analysis in Biology'. (Methuen and Co.: London.)

Mather, K. (1963). 'The Measurement of Linkage in Heredity'. (Methuen's Monographs in Biological Subjects.) pp. 44–68. (Methuen and Co.: London.)

McIntosh, R. A., and Baker, E. P. (1966). Differential reactions to three strains of wheat powdery mildew. *Aust. J. Biol. Sci.* **19**, 767–73.

Person, C. (1956). Some aspects of monosomic wheat breeding. *Can. J. Bot.* **34**, 60–70.

Pettigrew, Rona, and Driscoll, C. J. (1970). Cytogenetic studies of a chlorophyll mutant of hexaploid wheat. *Heredity* **25**, 650–5.

Pugsley, A. T., and Carter, M. V. (1953). Resistance to twelve varieties of *Triticum vulgare* to *Erysiphe graminis tritici*. *Aust. J. Biol. Sci.* **6**, 335–46.

Sears, E. R., and Briggle, L. W. (1969). Mapping the gene *Pm1* for resistance to *Erysiphe graminis* f. sp. *tritici* on chromosome 7A of wheat. *Crop Sci.* **9**, 96–7.

Sears, L. M. S., and Sears, E. R. (1968). The mutant *chlorina*-1 and Hermsen's virescent. Proc. 3rd Int. Wheat Genet. Symp., Canberra, Australia. pp. 299–304.

The, T. T. (1973). Chromosome location of genes conditioning stem rust resistance transferred from diploid to hexaploid wheat. *Nat. New Biol.* **241**, 256.

Waterhouse, W. L. (1930). Australian rust studies. III. Initial results of breeding for rust resistance. *Proc. Linn. Soc. N.S.W.* **55**, 596–636.

Watson, I. A., and Baker, E. P. (1943). Linkage of resistance to *Erysiphe graminis tritici* and *Puccinia triticina* in certain varieties of *Triticum vulgare*. Vill. *Proc. Linn. Soc. N.S.W.* **68**, 150–2.

Watson, I. A., and Luig, N. H. (1961). Leaf rust on wheat in Australia: a systematic scheme for the classification of strains. *Proc. Linn. Soc. N.S.W.* **86**, 241–50.

Watson, I. A., and Luig, N. H. (1963). The classification of *P. graminis tritici* in relation to breeding resistant varieties. *Proc. Linn. Soc. N.S.W.* **88**, 253–8.

Watson, I. A., and Luig, N. H. (1966). *Sr15*—a new gene for use in the classification of *P. graminis tritici*. *Euphytica* **15**, 239–50.

Appendix 1

Assume a model with the following characteristics:

(i) two linked loci A and B with a recombination value r;

(ii) female gametic transmissions normal;

(iii) differential male gametic transmission for the allele a—let this rate be k. Two values of k were used: $k = 0{\cdot}5$ which denotes a normal male trans-

mission, and $k = 0{\cdot}7$ which denotes higher male transmission for the allele *a* (male transmission rate estimated for *sr22*).

The expected gametic and zygotic frequencies from a double heterozygote when the two genes are in either coupling or repulsion are expressed in terms of k and r and are listed in Table 4. Recombination values are based on the above assumptions.

Table 4. Expected gametic and zygotic frequencies from a double heterozygote for coupling and repulsion phases

Phenotype	Gametic transmission		Expected F_2 frequency		
	Female	Male	General expression	Normal male transmission, $k = 0{\cdot}5$	Abnormal male transmission, $k = 0{\cdot}7$
			Coupling phase		
AB	$\frac{1}{2}(1-r)$	$(1-k)(1-r)$	$1-\frac{1}{2}k-\frac{1}{2}r+\frac{1}{2}kr^2$	$\frac{1}{4}(3-2r+r^2)$	$0{\cdot}65-0{\cdot}5r+0{\cdot}35r^2$
Ab	$\frac{1}{2}r$	$(1-k)r$	$\frac{1}{2}r-\frac{1}{2}kr^2$	$\frac{1}{4}(2r-r^2)$	$0{\cdot}5r-0{\cdot}35r^2$
aB	$\frac{1}{2}r$	kr	$kr-\frac{1}{2}kr^2$	$\frac{1}{4}(2r-r^2)$	$0{\cdot}7r-0{\cdot}35r^2$
ab	$\frac{1}{2}(1-r)$	$k(1-r)$	$\frac{1}{2}k-kr+\frac{1}{2}kr^2$	$\frac{1}{4}(1-2r+r^2)$	$0{\cdot}35-0{\cdot}7r+0{\cdot}35r^2$
Recombination value (%) between *Sr22* and *cn-A1*				$1{\cdot}9\pm0{\cdot}6$	$1{\cdot}8\pm0{\cdot}5$
			Repulsion phase		
AB	$\frac{1}{2}r$	$(1-k)r$	$\frac{1}{2}+\frac{1}{2}r-kr+\frac{1}{2}kr^2$	$\frac{1}{4}(2+r^2)$	$0{\cdot}5-0{\cdot}2r+0{\cdot}35r^2$
Ab	$\frac{1}{2}(1-r)$	$(1-k)(1-r)$	$\frac{1}{2}-\frac{1}{2}k-\frac{1}{2}r+kr-\frac{1}{2}kr^2$	$\frac{1}{4}(1-r^2)$	$0{\cdot}15+0{\cdot}2r-0{\cdot}35r^2$
aB	$\frac{1}{2}(1-r)$	$k(1-r)$	$\frac{1}{2}k-\frac{1}{2}kr^2$	$\frac{1}{4}(1-r^2)$	$0{\cdot}35-0{\cdot}35r^2$
ab	$\frac{1}{2}r$	kr	$\frac{1}{2}kr^2$	$\frac{1}{4}r^2$	$0{\cdot}35r^2$
Recombination value (%) between *Sr22* and *Pm1*				$40{\cdot}7\pm3{\cdot}3$	$40{\cdot}6\pm3{\cdot}1$
			cn-A1 and *Pm1*	$40{\cdot}2\pm3{\cdot}3$	$40{\cdot}1\pm3{\cdot}1$

Manuscript received 11 March 1975