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CYTOGENETICAL STUDIES IN WHEAT

X. MONOSOMIC ANALYSIS AND LINKAGE STUDIES INVOLVING GENES FOR RESISTANCE TO *PUCCINIA GRAMINIS* F. SP. *TRITICI* IN CULTIVAR KOTA

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SUMMARY

Two genes for resistance to *Puccinia graminis* f. sp. *tritici* were located in chromosome 2B of Kota wheat. Both were mapped in the long arm. The first designated *Sr28* was situated distally to *Sr9* and showed 34·6±2·8 per cent recombination with the centromere. The second gene, which was not definitely distinguished from *Sr16* either on the basis of recombination or by its response to several pathogen cultures, was inherited independently of the 2B centromere and of *Sr9*, but showed 38·2±1·9 and 29·2±4·2 per cent recombination with *Sr28*. Comparisons of present results with those from earlier studies suggested that Kota possesses at least five genes for resistance.

1. INTRODUCTION

THE common wheat, *Triticum aestivum* L., cultivar Kota was one of 12 *Triticum* genotypes chosen by Stakman and Levine to distinguish " races " of asexually propagating isolates of the stem rust pathogen, *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & Henn. (Stakman *et al.*, 1962). Following the work of Flor (1956) with flax rust involving the pathogen *Melampsora lini* (Ehrenb.) Lev. and the host *Linum usitatissimum* L., and similar work in several other disease systems (see Person, 1959), it can be assumed that any incompatibility in disease response, distinguishable from a standard compatibility, indicates that the particular host genotype possesses an allele(s) for resistance and that the particular pathogen isolate carries a corresponding allele(s) for avirulence. A study of a cross between such a " resistant " host and a " susceptible " one using the same pathogen isolate will indicate the number and inheritance of the host genes involved. Likewise, study of a cross between the " avirulent " pathogen isolate and a " virulent " one using the resistant host as tester will indicate the number and inheritance of genes concerned in the pathogen. A knowledge of host genotype permits deduction of pathogen phenotype, or genotype if the pathogen is haploid; and a knowledge of pathogen phenotype permits deduction of host phenotype, or genotype if the host is homozygous. Three criteria are useful in identifying resistance genes in the wheat stem rust system: firstly, formal genetic analysis of the host; secondly, tests with an array of pathogen isolates of known phenotypes; thirdly, the characteristics of the host pathogen interaction, the infection type. Infection types (Stakman *et al.*, 1962) are described on a " 0 " to " 4 " scale with " 0 " representing immunity, or no visible symptoms, and " 1 " to " 4 " representing infection types with sporulation, with " 4 " representing compatibility. The symbols " ; " for macroscopic hypersensitive flecks, " X " indicating a mesothetic or mixed

reaction, and " + " and " – " representing " more " or " less ", respectively, are also used.

From a study of F_2 cultures derived from a cross of two contrasting *P. graminis* f. sp. *tritici* parent cultures, Loegering and Powers (1962) hypothesised that Kota seedlings possessed two, and possibly three, genes for resistance. Berg *et al.* (1963) conducted a genetic analysis of Kota and identified three genes which they designated *SrKta1*, *SrKta2* and *SrKta3*. The present paper describes genetic and cytogenetic analyses of Kota and certain hybrid derivatives, and relates the results to those obtained by earlier workers. The methods employed included conventional pedigree analysis, monosomic analysis (Sears, 1953), telocentric mapping (Sears, 1966) and use of the second and third criteria described above.

2. Materials and methods

Kota W4 (W numbers refer to the Sydney University Wheat Accession Register) of Russian origin, was introduced about 1920 to the United States of America where it was commercially grown on a limited scale as a rust resistant wheat. A derivative cultivar, Ceres, was more widely grown until it was devastated by stem rust in 1935.

The following experiments were conducted:

(i) *Monosomic analysis*

Kota was crossed with 18 of the 21 monosomics of cultivar Chinese Spring originally supplied by Dr E. R. Sears, University of Missouri, U.S.A. F_2 seedlings derived from two monosomic plants in each cross were inoculated with *P. graminis tritici* culture 56-E1, a yellow-urediospore culture of North American origin. When infected with this culture, Kota produced infection type " 0; " whereas Chinese Spring produced infection type " 4 ".

(ii) *Confirmation of chromosome location and telocentric mapping*

Small samples of F_2 segregates of known infection types from each monosomic cross were transplanted and grown to maturity. In the crosses involving monosomics 1A and 2B, additional crosses and studies, summarised in fig. 1, were performed to confirm gene locations and to determine linkages with the centromere. The Chinese Spring ditelosomic-2BL stock was originally supplied by Dr E. R. Sears.

(iii) *Linkage studies*

Two backcross selections possessing genes derived from Kota (W2691*5/Kota) were supplied by Dr N. H. Luig of this Institute. Line AD had been selected for its infection type " ; " and Line AE for its infection type " 2 " when infected with culture 56-E1. Both lines were crossed with the chromosome substitution line, Chinese Spring (Kenya Farmer 2B), possessing the genetically linked alleles *Sr9b* and *Lr23* on opposite arms of chromosome 2B (McIntosh and Dyck, 1975). Additionally, an F_2 plant with infection type " 2 " from Chinese Spring monosomic-1A/Kota was crossed

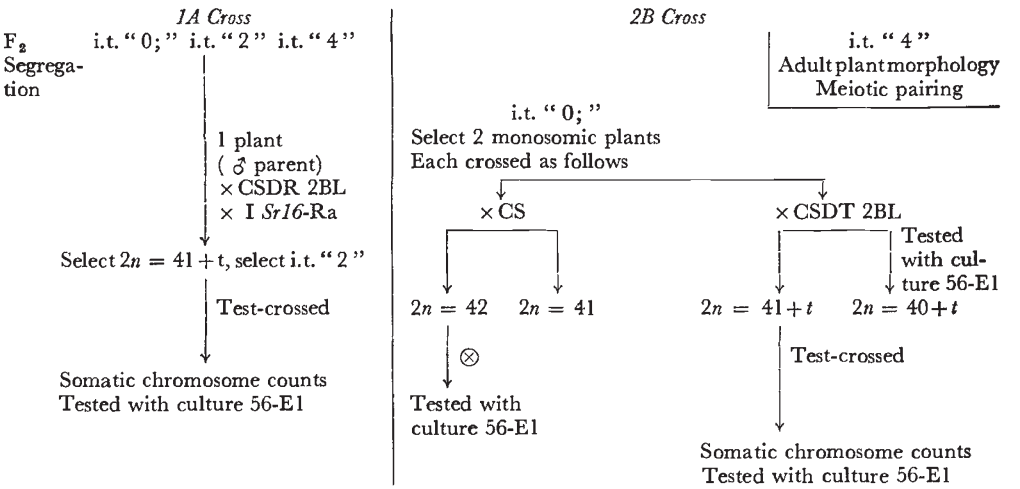


FIG. 1.—Crossing and testing procedures involved in confirming chromosome location and telocentric mapping of genes in Kota. i.t. = infection type; CSDT 2BL = Chinese Spring ditelosomic-2BL.

with I*Sr16*-Ra, W3416, a Chinese Spring line carrying *Sr16* and provided by Dr W. Q. Loegering, University of Missouri, U.S.A.

Linkage values were calculated from backcross data or were estimated by the method of maximum likelihood using F_2 phenotypic frequencies or F_2 genotypic frequencies determined from progeny tests. The various formulas for determining linkage values, and their standard errors, were given by Allard (1956).

(iv) *Comparative infection type studies*

Infection types produced by Kota, Line AD and Line AE, with a range of cultures were compared with those produced by several additional selections including four lines, Kota-A, Kota-B, Kota-C and Kota-D, derived from the study by Berg *et al.* (1963). Kota-A, Kota-B and Kota-C possess genes *SrKta1*, *SrKta2* and *SrKta3*, respectively. Chinese Spring W1806, Little Club W2 and W2691 were included as compatible standards.

(v) *Cultures of* P. graminis *f. sp.* tritici *and* P. recondita *Rob. ex Desm.*

In addition to culture 56-E1, other cultures of *P. graminis* f. sp. *tritici* were:

(1) 59-51A which is strain 59-ANZ-5,7 on the classification scheme of Watson and Luig (1963, 1966) and Luig and Watson (1977).
(2) 334, strain 126-ANZ-6, 7, 11.
(3) 69822, strain 326-ANZ-1, 2, 3, 5, 6.
(4) 111 × 36-F_1, an F_1 hybrid produced by Loegering and Powers (1962).

In order to classify genotypes for *Lr23* and *lr23* in crosses where Chinese Spring (Kenya Farmer 2B) was involved, *P. recondita* culture 67028, strain 26-ANZ-1, 3 (Watson and Luig, 1961), was used.

(vi) *Laboratory procedures*

Seedling populations for infection studies were sown at up to 25 plants per 9-cm pot. Inoculations were achieved by transfer of urediospores from previously infected seedlings to test populations with a scalpel, or by spraying with suspensions of urediospores in light mineral oil. After inoculation seedlings were maintained overnight in moist conditions to permit germination of urediospores, before being spaced on greenhouse benches for 12-14 days prior to scoring.

Somatic chromosome counts were made in root-tip squashes after treatment of excised root-tips in ice-water for 24 hours, fixation and staining by the Feulgen procedure. Meiotic examinations were made in squashes of selected anthers, fixed and prepared by a similar staining procedure.

3. Results

(i) *Monosomic analysis*

The segregation frequencies, pooled for each chromosome, are listed in table 1. The results suggest that two genes were segregating, one producing

Table 1

F_2 segregation in infection type classes for progenies of monosomic F_1 plants from crosses of 18 Chinese Spring monosomics with Kota when inoculated with culture 56-E1.

Chromosome involved	Infection type class		
	;	2	4
1A	68	8	9
1B	66	14	14
1D	170	36	18
2A	108	21	17
2B	111	—	26
2D	90	23	11
3B	95	22	16
4A	108	23	10
4B	113	18	17
4D	56	9	11
5A	122	36	9
5B	106	26	18
5D	35	6	2
6B	78	11	5
6D	95	18	18
7A	116	20	15
7B	114	23	16
7D	109	29	6
Total (excluding 2B)	1649	343	212

Using total values (2B excluded).

χ^2 contingency table		= 37·54	P > 0·3
χ^2 12 : 3 : 1 joint segregation		= 51·97	P < 0·001
χ^2 3 : 1 " ; " *v* remainder	1649 : 555	= 0·04	P > 0·8
χ^2 3 : 1 " 2 " *v* " 4 "	343 : 212	= 51·56	P < 0·001

infection type " ; " and the other, " 2 ". Segregation in the monosomic-2B population was distinctive in that no segregant with infection type " 2 " was observed, but there was a relatively high frequency (19·0 per cent) of seedlings with infection type " 4 ". This suggested either that the Kota gametes contributing to the cross with monosomic-2B were atypical and did not possess one of the genes ($\chi^2_{3:1}$ 111 : 26 = 2·65, $P_{1\ d.f.} > 0{\cdot}1$), or that both genes were situated in chromosome 2B and, since no recombination was possible in the monosome, the epistatic infection type " ; " was the only incompatible response expressed. According to the latter hypothesis plants with infection type " 4 " should be nullisomic. Although this implies a high nullisomic frequency, the morphology of the susceptible seedlings was consistent with nullisomy.

After exclusion of the data from the chromosome 2B cross, a contingency table chi-square analysis (table 1) indicated that the results for the other 17 chromosomes could be pooled. On the assumption of two segregating loci with the allele producing infection type " ; " epistatic to that producing " 2 ", there was a significant deviation ($P < 0{\cdot}01$) from an hypothesised ratio of 12 : 3 : 1. However the deviation was almost completely attributable to an increase in the frequency of seedlings with infection type " 4 " at the expense of those with infection type " 2 " and such a result would be expected if the two genes were linked in coupling. On this assumption a linkage value of $0{\cdot}382 \pm 0{\cdot}019$ was estimated by the method of maximum likelihood.

(ii) *Confirmation of gene location*

When random seedlings from the two infection type classes in the monosomic-2B cross were transplanted and investigated as described in fig. 1, all eight plants grown in the infection type " 4 " group were later in maturity than those in the infection type " ; " group. Six of the plants lacked the normal waxy stem but two were waxy. Waxlessness and lateness are distinctive characteristics of nullisomic-2B plants. On meiotic examination both waxy plants displayed 20 bivalents and an unpaired telocentric chromosome which was presumed to be 2BS since this arm is known to possess a wax-determining locus (Driscoll, 1966). It was concluded, therefore, that the gene producing infection type " ; " is located in chromosome 2BL. Furthermore, if the hypothesised gene producing infection type " 2 " was present in the 2B population it, also, must be located in chromosome 2BL.

When two monosomic F_2 plants with infection type " ; " were crossed as female parents with euploid Chinese Spring and with Chinese Spring ditelocentric-2BL plants, progenies possessing $2n = 42$ or $2n = 41 + t$ (t = telocentric chromosome) produced infection type " ; " whereas those with $2n = 41$ or $2n = 40 + t$ produced infection type " 4 ". This confirmed that the resistance allele was located in the unpaired chromosomes of the female parents. Since plants with $2n = 40 + t$ displayed 20 bivalents and a telocentric univalent at meiosis, parent plants must have been monosomic for 2B. Monotelodisomic ($2n = 41 + t$) sibs were test-crossed for telocentric mapping.

When selfed seedlings derived from euploid hybrids were tested with culture 56-E1, they segregated for infection types " ; ", " 2 " and " 4 ",

confirming that the Kota gametes in the original monosomic-2B cross had carried the second gene.

(iii) *Telocentric mapping*

In his original description of telocentric mapping Sears (1966) took advantage of the knowledge that if a dominant marker is located in the full chromosome and heterozygous monotelodisomic plants are used as male parents then for an estimate of linkage with the centromere, cytological study of the test-cross population can be limited to those individuals with the recessive phenotype. Because of certation of male gametes possessing the telosome, the majority of test-cross products will be euploid, and if linkage with the centromere is displayed, or if the locus under investigation is located in the non-paired arm, then relatively few plants will exhibit the recessive phenotype. Linkage is estimated from the proportion of such individuals that are euploid. The present author prefers to cytologically examine all test-cross individuals and to maximise the possibility of recovering recombination products possessing telocentric chromosomes carrying the dominant allele so that marked telocentric lines (Baker and McIntosh, 1973) can be established for future investigations. Hence monotelodisomic heterozygotes can be test-crossed in either direction. For this investigation these were test-crossed in both directions; the following pooled results were obtained when progenies were infected with culture 56-E1.

Somatic chromosome number	Infection type			
	" ; "	" 2 "	" 4 "	
$2n = 42$	118	17	39	174
$2n = 41+t$	41	19	46	106
	159	36	85	280

For estimating the genetic distance of the epistate factor from the centromere, recombinants included those euploid plants not producing infection type " ; " and those monotelodisomics which did produce " ; ". That is, recombination is $\frac{17+39+41}{280} = 0{\cdot}346 \pm 0{\cdot}028$.

Since a considerable number of test-cross individuals in both chromosome classes produced infection type " 2 " and considering the linkage value of 0·382 estimated from the data in table 1 the second gene must be located distally from the centromere. A further estimate of linkage between the two genes is provided by the proportion of plants with infection type " 2 " among those not having infection type " ; ", *i.e.* $\frac{17+19}{121} = 0{\cdot}298 \pm 0{\cdot}042$. This estimate is not significantly different from the previous estimate of 0·382 ($P > 0{\cdot}05$).

An estimate of linkage between the second locus and the centromere was obtained from test crosses of two monotelodisomic plants with infection type " 2 " from the cross Chinese Spring ditelosomic-2BL with a random

plant with infection type " 2 " in the cross Chinese Spring monosomic-1A/Kota (fig. 1). The following pooled results were obtained:

Somatic chromosome number	Infection type " 2 "	Infection type " 4 "	
$2n = 42$	31	28	59
$2n = 41 + t$	23	15	38
	54	43	97

Linkage between the centromere and the second locus was estimated to be $\frac{23+28}{97} = 0{\cdot}529 \pm 0{\cdot}051$.

(iv) *Conventional linkage studies*

Line AD was assumed to carry the allele *A* determining infection type " ; " and Line AE was assumed to carry the allele *B* determining infection type " 2 ".

(a) *Line AD/Chinese Spring (Kenya Farmer 2B)*

Table 2 lists the F_2 genotypic frequencies as determined by separate inoculations of F_3 progenies with *P. recondita* culture 67028 for classification of *Lr23* and *lr23*, and *P. graminis tritici* cultures 334 for classification of *Sr9b* and *sr9b* and 59-51A for *A* and *a*. Although culture 59-51A was also

TABLE 2

Genotypic frequencies from the cross involving Line AD (genotype AA*) and Chinese Spring (Kenya Farmer 2B)* (Lr23 Lr23 Sr9b Sr9b)

Lr23 Lr23 Sr9b Sr9b AA	—	*Lr23 lr23 Sr9b Sr9b AA*	—	*lr23 lr23 Sr9b Sr9b AA*	1	1		
Aa	3	*Aa*	4	*Aa*	3	10	43	
aa	21	*aa*	9	*aa*	2	32		
Sr9b sr9b AA	2	*Sr9b sr9b AA*	12	*Sr9b sr9b AA*	4	18		
Aa	11	*Aa*	51	*Aa*	6	68	102	
aa	4	*aa*	9	*aa*	3	16		
sr9b sr9b AA	—	*sr9b sr9b AA*	18	*sr9b sr9b AA*	20	38		
Aa	—	*Aa*	4	*Aa*	9	13	52	
aa	—	*aa*	—	*aa*	1	1		
	41		107		49	197		

	χ^2 1 : 2 : 1	P-value
Lr23 Lr23 : *Lr23 lr23* : *lr23 lr23* = 41 : 107 : 49	2·12	>0·3
Sr9b Sr9b : *Sr9b sr9b* : *sr9b sr9b* = 43 : 102 : 52	1·07	>0·5
AA : *Aa* : *aa* = 57 : 91 : 49	1·79	>0·3

avirulent for *Sr9b* the infection type " ; " produced by *A* was epistatic and distinctive to that produced by *Sr9b*. Since culture 334 was virulent on Line AD complete classification of genotypes was possible. Segregation at each locus was in accordance with hypothesised 1 : 2 : 1 ratios (table 2); however, the joint segregations for pairs of genes indicated linkage in each

instance. The following recombination values were estimated by the method of maximum likelihood:

Lr23-Sr9b	$0{\cdot}223 \pm 0{\cdot}024$
Lr23-A	$0{\cdot}285 \pm 0{\cdot}028$
Sr9b-A	$0{\cdot}168 \pm 0{\cdot}021$

Clearly, *Sr9b* is situated between *Lr23* and *A*. In terms of genetic map distance, the recombination value for *Lr23* and *A* can be corrected for double crossing-over, *i.e.* $0{\cdot}285 + 2.0{\cdot}223.0{\cdot}168 = 36$ map units. This value is similar to the 39·1 obtained when the separate values are summed.

(b) *Line AE/Chinese Spring (Kenya Farmer 2B)*

Frequencies of various F_2 phenotypic and genotypic classifications are listed in table 3. Again, the same three cultures were used to test F_3 families. Culture 334 is virulent for gene *B* and hence permitted classification for *Sr9b* and *sr9b*. However, culture 59-51A is avirulent for both *Sr9b* and *B* and, since both factors produced similar infection types, they could

TABLE 3

Genotypic frequencies from the cross involving Line AE (BB) *and Chinese Spring (Kenya Farmer 2B)* (Lr23 Lr23 Sr9b Sr9b)

Lr23 Lr23 Sr9b Sr9b	—	27	*Lr23 lr23 Sr9b Sr9b*	—	24	*lr23 lr23 Sr9b Sr9b*	—	5	56
Sr9b sr9b	*B*—	16	*Sr9b sr9b*	*B*—	82	*Sr9b sr9b*	*B*—	22	120
	bb	3		*bb*	23		*bb*	5	31
sr9b sr9b	*BB*	1	*sr9b sr9b*	*BB*	6	*sr9b sr9b*	*BB*	11	18
	Bb	1		*Bb*	14		*Bb*	16	31
	bb	—		*bb*	9		*bb*	19	18
		48			158			68	274

	χ^2	P-value
Lr23 Lr23 : *Lr23 lr23* : *lr23 lr23* 48 : 158 : 68	9·36*	<0·01
Sr9b Sr9b : *Sr9b sr9b* : *sr9b sr9b* 56 : 151 : 67	3·75*	>0·1
BB : *Bb* : *bb* 18 : 31 : 18	0·37*	>0·8
Joint segregation *Sr9b* and *B* 56 : 120 : 31 : 13 : 31 : 18	5·89**	>0·3

* Ratio tested 1 : 2 : 1; ** Ratio tested 4 : 6 : 2 : 1 : 2 : 1.

not be distinguished with this culture. Because of insufficient seed numbers it was not always possible to distinguish families segregating for both genes from those segregating for *Sr9b* and *sr9b*, but homozygous *BB*. Therefore, complete classification for *B* and *b* was possible only in the absence of *Sr9b*.

Segregation for *Lr23* and *lr23* was not in accordance with expectation for a single locus; there was a deficiency of *Lr23* homozygotes and a corresponding excess of heterozygotes. However, segregations of *Sr9b sr9b* genotypes conformed with the expected 1 : 2 : 1 ratios. A linkage value of $0{\cdot}241 \pm 0{\cdot}022$ was estimated from the observed frequencies for *Lr23* and *Sr9b*. This estimate was similar to the corresponding value in the Line AD cross. The chi-square value for joint segregation of *Sr9b* and *B* indicated that these genes could be genetically independent.

(c) *Chinese Spring monosomic-1A/Kota, F_2/ I* Sr16-*Ra*

Duplicate F_3 populations from 48 F_2 plants were inoculated with cultures 56-E1 and 59-51A. All individuals in all populations, as well as

progeny of the parental F_2 selection and I*Sr16*-Ra, produced infection type " 2 " with both cultures indicating that the genes involved are closely linked, or allelic and possibly identical. If there are two genes at different loci then the probability of observing recombinant genotypes approximates $r-\frac{1}{4}r^2$ where r is the recombination value. Since no recombinant genotypes were observed among 48 F_2 families then the maximum value of r at P = 0.05 is given by

$$1-r+\tfrac{1}{4}r^2 = 0{\cdot}05^{1/48}$$
$$r = 0{\cdot}0616$$

(v) *Comparative infection type studies*

Table 4 lists the infection types produced when Kota, Kota derivatives and relevant controls were infected with four *P. graminis tritici* cultures. Kota produced infection type " 0; " with three cultures and " 2+ " with 69822, an Australian field isolate known to be avirulent for *Sr7b*. The similar responses of I*Sr7b*-Ra, Kota, Ceres, Marquis and Kota-A indicate they

TABLE 4

Infection types produced by Kota and other wheats when tested with four P. graminis tritici *cultures*

Stock	Culture Strain	56-E1 —	59-51A 59-ANZ-5, 7	69822 326-ANZ-1, 2, 3, 5, 6	111 × 36—F_1 —
Kota		0;	0;	2+	0;
Ceres		0;	0;	2+	0;
Marquis		4	;1—	2+	0;
Line AD		;	;	4	0;
Line AE		2	2	4	2—
W 2691		4	4	4	4
Kota-A		;	;	2+	0;
-B		4	4	4	;1—
-C		4	4	4	2—
-D		4	4	4	3
Little Club		4	4	4	3+
I *Sr7b*-Ra		3	2+	2+	3
I *Sr16*-Ra		2	2	3+	3
Chinese Spring (Hope 1D)		4	4	4	;1
Chinese Spring		4	4	4	3

carry *Sr7b* in common. Ceres wheat was produced from a cross between Kota and Marquis and results indicate that Ceres inherited at least one resistance gene from Kota. This gene is present in Line AD and Kota-A and is the gene located and designated *A* in this study.

The results do not show whether gene *B* is present in Ceres. As far as cultures 56-E1 and 59-51A are concerned, Line AE possesses gene *B*, which from genetic results, is closely linked or allelic with *Sr16*. However, if *B* is identical with *Sr16* then Line AE must carry a second gene for resistance to culture $111 \times 36-F_1$ which is virulent for *Sr16*. Such a gene could be

identical with that present in Kota-C. Alternatively, if gene *B* in Line AE confers resistance to culture $111 \times 36 - F_1$, as well as to 56-E1 and 59-51A, then it must be different from *Sr16* and from the gene in Kota-C. Kota-B was resistant to $111 \times 36 - F_1$, but the gene involved appeared to be identical with *Sr18* carried by the chromosome substitution line, Chinese Spring (Hope 1D).

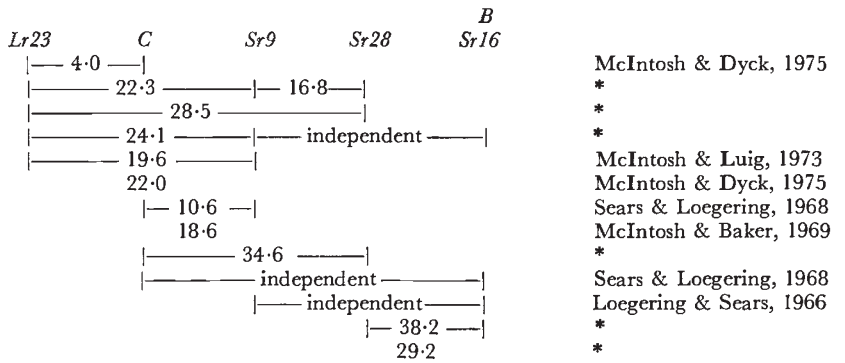
4. Discussion

The present study shows clearly the location in Kota wheat of two genes for resistance to *P. graminis* f. sp. *tritici* in chromosome 2B. The first, designated herein as *A* and also present in Ceres, Line AD and Kota-A, is situated distally to *Sr9* and represents a newly defined locus. The symbol *Sr28* has been allocated to this gene. The second gene, *B*, present in Line AE, did not recombine with *Sr16*, but if *B* is located at a separate locus it is within six cross-over units of *Sr16* ($P > 0{\cdot}05$). Obviously, gene *B* could be allelic with, but distinctive from, *Sr16*. However, if *B* is *Sr16*, then Line AE must possess a second gene conferring resistance to culture $111 \times 36 - F_1$ and possibly the same as that present in Kota-C. Further studies involving I*Sr16*-Ra, Line AE and Kota-C are necessary to resolve these alternative hypotheses.

Working with F_2 cultures of the pathogen, Loegering and Powers (1962) obtained evidence for at least two, and possibly three, genes for resistance in Kota. One of these was undoubtedly *Sr28* and another, *Sr18*. On the other hand Berg *et al.* (1963) identified three genes, *viz. SrKta1* corresponding to *Sr28*, *SrKta2* corresponding to *Sr18* and *SrKta3* which is distinctive. It is now clear, however, that culture 111-SS2 used by Berg *et al.* was virulent for *Sr7b* and presumably for gene *B* identified in the present studies. Hence Kota appears to possess at least five genes for resistance to *P. graminis* f. sp. *tritici*, *viz. Sr7b*, *Sr18*, *Sr28*, *B* and *SrKta3*. If *B* proves to be *Sr16* then Line AE may carry *SrKta3* as well as *Sr16*, despite the fact that four backcrosses to W2691 were performed in its production.

Using Kota for race identification in *P. graminis* f. sp. *tritici*, any of five genes could be responsible for incompatibility with the pathogen. However, many field variants important in agriculture must possess all five corresponding genes for virulence, since they are virulent on Kota seedlings. The loss of resistance in Ceres wheat in North America was caused by the increase of race 56 which is virulent for *Sr28* as well as the other genes in Kota. The most frequent pathogen variants with avirulence on Kota seedlings are avirulent on seedlings possessing only *Sr7b* and are avirulent also on Marquis, another of the standard genotypes used in race identification (Stakman *et al.*, 1962). Many genes, such as *Sr18* which may be very common in the host population (Baker *et al.*, 1970), are seldom identified in genetic studies because the experimental test cultures of the pathogen have the corresponding genes for virulence.

The *Lr23-Sr16* region of chromosome 2B has probably undergone more recombination studies than any other region of the wheat genome. The various recombination estimates from this and earlier studies involving four loci for disease reaction and the centromere are shown in fig. 2. The linkage values are generally additive and there is no evidence for genetic interference significantly affecting the recombination values.



* Estimates obtained in this study.

FIG. 2.—Linkage estimates (%) involving the *Lr23-Sr16* region of chromosome 2B. C = centromere.

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