

LINKAGE STUDIES OF MUTANTS IN MAIZE WITH PIGMENT DEFICIENCIES IN ENDOSPERM AND SEEDLING¹

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THIS paper will be concerned mainly with those pigment defective mutants of maize that have pale yellow or white endosperms and chalky-white albino or pale green seedlings. Most early workers with chlorophyll deficient mutants of maize report only on seedling phenotype and do not mention the color of the endosperm. The first report of albino mutants which are also deficient for carotenoids in the endosperm are those of LINDSTROM (1923), EYSTER (1924a,b, 1931) and MANGELSDORF (1923, 1926, 1930). The latter two workers were primarily concerned with studying viviparous mutants which in most instances also have white endosperms and albino seedlings. More recent reports of genetic work with these mutants have been made by EVERETT (1949), TULPULE (1954), ROBERTSON (1955) and SIDHU (1960).

There have been reports in the literature of two mutants that have white endosperm and pale green seedlings. One of these was a description of an unnamed mutant by ANDERSON (1924). The other mutant, albescent, was first described by PHIPPS (1929), and was placed on chromosome 2 by PERRY and SPRAGUE (1936). GRANER and ACCORSI (1949) compared the development of chloroplasts in this mutant with those of a white endosperm albino. There is some confusion in the symbols used for the albescent mutant. The symbol γ_s was used for the white endosperm condition and *al* for the seedling phenotype. Early, it was felt that two closely linked genes were involved. However, the work of PERRY and SPRAGUE (1936) would suggest that the reported crossovers are best explained by heterofertilization.

Recent work by KOSKI and SMITH (1951), SMITH, DURHAM and WURSTER (1959), ANDERSON and ROBERTSON (1960), and ANDERSON (unpublished) has suggested that the normal alleles of these mutants are concerned with carotenoid production and that chlorophyll is only secondarily involved. The present evidence suggests that yellow carotenoids are necessary for the protection of chlorophyll from oxidative photodestruction (ANDERSON and ROBERTSON 1960). The mutant seedlings, most of which are completely devoid of yellow carotenoids, accumulate chlorophyll if grown under dim light. However, the chlorophyll is destroyed as fast as it is formed under normal growing conditions where light is plentiful.

The promising results obtained from the biochemical studies with these

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mutants by the above workers, as well as the more recent genetic studies by SIDHU (1960) and ROBERTSON (1960) would indicate that these pigment deficient mutants will be subject to further biochemical and genetic studies in the near future. Therefore, it is of value to have precise linkage information available. ROBERTSON (1955) presented linkage data that was then available for the viviparous albino mutants. Since that time, additional linkage data have been obtained which make possible the placing of these genes with more precision. These new data will be presented as well as linkage data for the nonviviparous albino mutants.

MATERIALS AND METHODS

Description of mutants

Because mutants of divergent phenotypes will be discussed, it is advisable to group them for convenience of reference. ROBERTSON (1955) suggested that these mutants be classified into three groups: (1) those with pale yellow or white seeds and albino viviparous seedlings, (2) those which show little if any tendency to germinate prematurely but are otherwise similar to Class 1, and (3) those which are similar to Class 1 in having pale yellow or white seeds which do not germinate prematurely but differ in that they produce pale green seedlings.

Subsequent allele tests with pastel-4889 and pastel-8686, both of which belong to Group 3 and have never shown any tendency to be viviparous, have indicated that they are allelic to the Group 1 mutants vp_8 and w_3 respectively. Further allele tests have created additional difficulties for establishing a satisfactory system of classification of these mutants. Pastel-8549, and white-mutable, Group 3 mutants, have been shown to be allelic to γ_1 (ROBERTSON and ANDERSON 1961), which is not a member of any of the three groups.

The results of these allele tests indicate that the classification suggested above is an artificial one, since it places allelic mutants in different groups. However, in spite of this, such a system of classification is useful for grouping mutants with similar phenotypes if it is understood that it is based solely on phenotype and in no way implies genotypic relationships. In order to incorporate the results of the allele tests, a further group will be added giving a total of four, as summarized in Table 1.

Different names and symbols have been used by various workers to designate these genes. No attempt will be made here to settle on a unifying system nomen-

TABLE 1

A summary of the phenotypes that characterize the groups of mutants discussed in this report

Group	Phenotype of endosperm	Phenotype of plant
1	white	albino-viviparous
2	white	albino
3	white	pale green (pastel)
4	white	green

clature. This cannot be done intelligently until more is known about the chemistry of these mutants and until their relationship to other pigment deficient mutants of corn is better understood.

The names and symbols used in this report will be those of the workers that first found and/or published on the particular gene under consideration. Where alleles are discussed, they will usually be designated by a name and/or symbol that implies their source (e.g., *W_{Everett No. 1}*, *W_{Smith}*, *W_{Sprague}*, and *W_{Brawn No. 1}*, etc.) Symbols that contain a four number subscript (e.g., *w₆₄₇₄*, *w₇₇₄₈*) stand for genes that were found in stocks derived from seed exposed to the Bikini atom bomb and other sources of irradiation that were first grown at the California Institute of Technology in Pasadena, California.

Source of genes

The sources of the genes *vp₂*, *vp₅*, *vp₉*, *ps*, and *w₃* are given by ROBERTSON (1955). Lemon-white-1, *lw₂*, *lw₃*, and *lw₄* were obtained from DR. M. M. RHOADES and their origin is discussed by TULPULÉ (1954). The *cl₁* mutant was obtained from DR. HERBERT L. EVERETT and its origin has been described by him (EVERETT 1949). Mutant *w₇₇₄₈* and its allele, *w₈₆₂₄*, were first found in stocks derived from irradiated seed grown at the California Institute of Technology. The origins of *pas₈₅₄₉* and *w_{mut}* are given by ROBERTSON and ANDERSON (1961). Seed of the albescens mutant was obtained from DR. GEORGE F. SPRAGUE.

Linkage techniques

Extensive use has been made of gene marked translocations and translocations with B-type chromosomes in locating genes in these studies. ANDERSON (1952, 1956) and ROBERTSON (1955) have described the principles involved in the use of gene marked translocations and ROMAN and ULLSTRUP (1951) have described the use of translocations with B-type chromosomes in locating genes.

Because albino plants do not survive beyond the seedling stage, it is impossible to obtain the necessary double and triple homozygotes for testcrossing. This necessitated the use of the modified testcross as described by ROBERTSON (1955).

Genetic studies

Viviparous-2: Allele tests have revealed that a similar mutant, green mosaic, which first appeared in the stocks at the California Institute of Technology, was allelic to *vp₂*. Both of these mutants belong to Group 1, but green mosaic differs from *vp₂* in that it is an unstable gene that regularly reverts to normal in both endosperm and seedling. Green mosaic seeds are white or pale yellow with spots of dark yellow tissue and the seedlings are albino with small streaks of normal green tissue. Very little is known about the nature of the mutable system in this mutant. The presence of mutable and stable white seeds and seedling on the same selfed ear indicates that an independent modifier is involved.

The data reported by ROBERTSON (1955) placed *vp₂* in the short arm of chromosome 5 to the left of *bm₁*. The results of the three point tests recorded in Table 2 indicate that *vp₂* is to the right of *a₂*. The *ps* mutant is another Group 1

TABLE 2

Summary of three point data from testcrosses involving vp_2

Genotype of F_1	Parental combinations		Recombinations						Percent recombinations		
			Region 1		Region 2		Region 1 and 2		Region 1	Region 2	
$a_2 \text{ } vp_2 \text{ } +$ $+ \text{ } + \text{ } ps$	167	171	3	2	1	2	1	0	347	1.7	1.2
$+ \text{ } + \text{ } bm_1$ $a_2 \text{ } vp_2 \text{ } +$	41	36	1	2	5	2	0	0	87	3.4	8.0

albino mutant (see below). Table 3 summarizes the linkage data for vp_2 and its nearest neighbors. These data indicate the following linkage relationship: $a_2-3.3-vp_2-0.9-ps-3.3 \text{ } bm_1$.

Viviparous-5: The following five alleles of this Group 1 mutant have been found: w_{8605} and w_{K40} , $w_{Mumm \text{ No. } 1}$, $w_{Brawn \text{ No. } 1}$ and $w_{Sprague \text{ No. } 1}$. All of these alleles are also Group 1 mutants.

ROBERTSON (1955) placed this gene in the short arm of chromosome 1 to the left of *P*. The data from Table 4 permit the placing of this gene with some precision. It will be noted that the data included in this table were obtained from a self. This was made necessary because a series of unfortunate coincidences prevented obtaining the necessary testcrosses. Non- vp_5 seeds from selfed ears of plants of the genotype $T1-2c \text{ } sr \text{ } +/+ \text{ } + vp_5$ were planted and the resulting plants classified for sterility and striate and then self pollinated. The ears from these F_2 plants were classified for vivipary and seedling tested to determine the striate constitution. With this information, it is possible to determine the genotype of the nonviviparous gametes that gave rise to the F_2 plants. Viviparous classes cannot be used because it is not possible to classify for striate when vp_5

TABLE 3

Summary of two point data from testcrosses involving vp_2

Genotype of F_1	Parental combinations		Recombinations		Totals	Total recombinations	Percent recombinations
$a_2 \text{ } +$ $+ \text{ } vp_2$	132	137	9	6	284	15	5.3
$+ \text{ } +$ $a_2 \text{ } vp_2$	219	206	3	6	434	9	2.1
					718	24	3.3
$+ \text{ } ps$ $vp_2 \text{ } +$	208	218	3	1	430	4	0.9
$+ \text{ } bm_1$ $vp_2 \text{ } +$	182	209	6	11	408	17	4.2

TABLE 4

Non- vp_5 gametes functioning in self of $\frac{T1-2c\ sr\ +}{+ + vp_5}$

F ₁ parents	Parental combinations <i>T sr +</i>	Recombinations			Totals	Percent recombinations	
		Region 1 <i>+ sr +</i>	Region 2 <i>+ + +</i>	Region 1 and 2 <i>T + +</i>		Region 1	Region 2
58-5638-75	135	2	2	0	139	1.4	1.4
58-5640-14	138	0	1	0	139	—	0.7
58-5642-18	101	0	1	0	102	—	1.0
58-5643-14	115	3	0	0	118	2.5	—
Totals	489	5	4	0	498	1.0	0.8

is present. It is thought that these results are very reliable since very little crossing over is observed in either regions 1 or 2. The data from this table indicate an order of *T1-2c-sr-vp₅*.

Table 5 gives linkage data for *vp₅* with *ts₂* and *P*. Backcross data reported by EMERSON (1939) and ANDERSON (1941) indicate that *ts₂* is about one unit to the left of *P*. The data from Table 5 would indicate that the order of these two genes might be reversed. However, since the numbers are small, especially in the case of the linkage with *P*, the discrepancy here might be the result of sampling error. Assuming that *ts₂* is the left most gene, the present data indicate the following linkage relationship for *vp₅*: *T1-2c-1.0-sr-0.8-vp₅-23.4-ts₂*. Since *T1-2c* has been placed by LONGLEY (1958) in the short arm of chromosome 1 at .77, this would mean that *sr* and *vp₅* are proximal to this break point. It must be kept in mind that the presence of *T1-2c* in heterozygous condition in the F₁ may have reduced the true crossover value for the *T-sr* and *sr-vp₅* regions.

Viviparous-9: Two alleles to this Group 1 mutant have been found: *Pastel₄₈₈₉*, a Group 3 mutant; and *w_{Brawn No. 2}*, a Group 1 mutant.

ROBERTSON (1955) indicated that this gene was in the long arm of chromosome 7 and that the most likely order was *T7-9a-gl₁-vp₉*. This order was based on two point tests and assumed the then reported position of L.29 for the break point of *T7-9a* in the long arm of chromosome 7. Since then three point tests have been made (Table 6) and a corrected estimate of the break point as 7 L.63 has been published (LONGLEY 1958). Using this additional information and the linkage data from Table 7, the linkage relationships for *vp₉* can be summarized as follows:

TABLE 5

Summary of two point data from testcrosses involving vp_5

Genotype of F ₁	Parental combinations		Recombinations		Totals	Total recombinations	Percent recombinations
$\frac{+ ts_2}{vp_5 +}$	122	107	40	30	299	70	23.4
$\frac{+ P}{vp_5 +}$	75	69	26	15	185	41	22.2

in the neighborhood of *bm*₁. Although the data from Table 8 will permit the placement of *ps* with respect to *pr*, they are not so helpful in placing the gene with respect to *bm*₁. However, linkage results given in Tables 2 and 3 would suggest that *ps* is distal to *bm*₁ and proximal to *a*₂ and *vp*₂. Using the available information, the following linkage relationships are indicated: *a*₂—3.3—*vp*₂—0.9—*ps*—2.4—*bm*₁.

White-3: Two alleles of this Group 1 mutant have been found: *w*_{Everett No. 1}, a Group 1 mutant; and *Pastel*₈₆₈₆, a Group 3 mutant.

Because of linkage found with *T2-4c*, ROBERTSON (1955) had placed this gene on chromosome 2. The data from three point tests involving *v*₄ are given in Table 9. All of these are consistent in indicating that *w*₃ is to the right of *v*₄ in the long arm of chromosome 2. Table 10 (first cross) summarizes the linkage information available for *v*₄ and *w*₃ for those crosses where translocations were

TABLE 9

Summary of three point data from testcrosses involving *w*₃

Genotype of F ₁	Parental combinations		Recombinations						Percent recombinations		
			Region 1		Region 2		Region 1 and 2		Totals	Region 1	Region 2
$\frac{B v_4 +}{+ + w_3}$	63*	92	54	26*	17*	41	7	4*	304	29.9	22.7
$\frac{ts_1 v +}{+ + w_3}$	0*	37	10	4*	1*	16	2	2	72	25.0	29.2
$\frac{+ + T2-4c}{v_4 w_3 +}$	77	80	17	31	1	1	0	0	207	23.2	1.0
$\frac{+ + T2-9d}{v_4 w_3 +}$	96	65	15	18	0	1	0	0	195	16.9	0.5

* The numbers in these classes are low because of low viability of *v*₄ plants.

TABLE 10

Summary of two point data from testcrosses involving *w*₃

Genotype of F ₁	Parental combinations		Recombinations		Totals	Total recombinations	Percent recombinations
$\frac{v_4 +}{+ w_3}$	93*	193	24*	66	376	90	23.9
$\frac{+ T2-4c}{w_3 +}$	283	288	4	4	579	8	1.4
$\frac{+ Ch}{w_3 +}$	91	83	51	38	263	89	33.8

* The numbers in these classes are low because of low viability of *v*₄ plants.

not involved. These data indicate 23.9 percent recombination between these two loci. The three point tests with *T2-4c* and *T2-9d* indicate that these translocations are probably distal to w_s . LONGLEY (1958) has indicated the break points in chromosome 2 to be L.81 for *T2-4c* and L.83 for *T2-9d*. The similar linkage values with w_s observed for these two translocations agree with the cytological observation that their break points are very close together. A point test with chocolate (Table 10, third cross) gave 33.8 percent recombination with w_s . Since *Ch* is the most distal gene indicated by RHOADES (1954) on the long arm of chromosome 2, it is most likely that w_s is proximal to it. The data from Tables 9 and 10 indicate the following linkage relationship for w_s : v_s —23.9— w_s —1.2—“*T*”—32.6 *Ch*. Because the break points of *T2-4c* and *T2-9d* are so close, “*T*” in this summary is used to stand for both of them and the crossover value of 1.2 was obtained by combining the linkage data for the two translocations.

Lemon-white-1: TULPULE (1954) had located this Group 2 mutant on the long arm of chromosome 1 by the use of *TB-1a*. *White*₆₄₇₃, a Group 2 mutant, which had also been placed on chromosome 1 by linkage with *T1-4a*, proved to be allelic to lw_1 .

The limited three point data from Table 11 suggest that lw_1 is proximal to the break point of *T1-4a* which has been placed at 1L.51 (LONGLEY 1958). Table 12,

TABLE 11

Summary of three point data from a testcross involving lw_1

Genotype of F_1	Parental combinations		Recombinations				Percent recombinations				
			Region 1		Region 2		Totals	Region 1	Region 2		
$P + T1-4a$ $+ lw_1 +$	19	14	11	16	7	9	6	7	89	44.9	32.6

TABLE 12

Summary of two point data from testcrosses involving lw_1

Genotype of F_1	Parental combinations		Recombinations		Totals	Total recombinations	Percent recombinations
$+ T1-4a$ $lw_1 +$	99	67	36	32	234	68	29.1
$+ +$ $lw_1 T1-4a$	69	86	20	25	200	45	22.5
					434	113	26.0
$Kn +$ $+ lw_1$	234	292	0	6	532	6	1.1
$+ bm_2$ $lw_1 +$	58	51	16	18	143	34	23.8

(first and second crosses) summarizes the linkage tests with *T1-4a* and indicates that *lw₁* is 26.0 crossover units from the break point in chromosome 1. Linkage tests with *Kn* indicate very close linkage (Table 12, third cross). The figure of 1.1 percent recombination is probably a maximum value. It will be noticed that the crossovers are all in the ++ class. Since any seeds resulting from contamination would fall in this class, these may not represent actual crossovers. So far no crossovers of the *Kn lw₁* class have been observed. A recombination value with *bm₂* of 23.8 percent has been found (Table 11, fourth cross). The genetic studies with *lw₁* indicate the following linkage relationships: *Kn*-(1.1)-*lw₁*-23.8-*bm₂*. At present, it is impossible to place *lw₁* to the left or right of *Kn*.

Lemon-white-2: Using F₂ data TULPULE (1954) had placed *lw₂*, a Group 2 mutant, on chromosome 5. *White 7752*, which had been placed on chromosome 5 by linkage with *T5-9c*, and the unplaced genes *wEverett No. 3*, *wSmith No. 1*, *wBrunson*, *wChase* have proven to be allelic to *lw₂*. All these alleles are Group 2 mutants.

The results of three point tests with *pr* given in Table 13 (first and second crosses), while inconclusive, would suggest that *lw₂* is to the right of this gene. The summary of all linkage data with *pr* (Table 14, first cross) indicate 2.2 percent crossing over between this locus and *lw₂*. The three point tests with *ps*

TABLE 13

Summary of three point data from testcrosses involving *lw₂*

Genotype of F ₁	Parental combinations		Recombinations						Percent recombinations		
			Region 1		Region 2		Region 1 and 2		Totals	Region 1	Region 2
$\frac{+ pr +}{ps + lw_2}$	75	72	11	14	0	1	2	0	175	15.4	1.7
$\frac{bm_1 pr +}{+ + lw_2}$	71	78	19	26	1	3	0	1	199	23.1	2.5
$\frac{+ + T5-9a}{ps lw_2 +}$	71	46	12	41	8	22	2	6	208	29.3	18.3
$\frac{+ T5-9d +}{ps + lw_2}$	67	99	0	0	17	17	0	1	201	0.5	17.4

TABLE 14

Summary of two point data from testcrosses involving *lw₂*

Genotype of F ₁	Parental combinations		Recombinations		Totals	Total recombinations	Percent recombinations
$\frac{+ gl_s}{lw_2 +}$	207	202	0	0	409	0	0

and translocations *T5-9a* and *T5-9d* (Table 13, third and fourth crosses) indicate that *lw*₂ is proximal to the break point of the former translocation at 5L.69 (LONGLEY 1958) 18.3 units and distal to the break point of the latter at 5L.14 (LONGLEY 1958) 17.4 units. Linkage tests with *gl*₃ (Table 14, second cross) have revealed no crossing over in 409 plants tested.

The following is a summary of the available linkage information on *lw*₂: *T5-9d*—15.2—*pr*—2.2—*lw*₂*gl*₃—18.3—*T5-9a*.

Lemon-white-3 and *Lemon-white-4*: These duplicate genes are Group 2 mutants and were first described by TULPULE (1954). On the basis of F₂ data, TULPULE (1954) placed *lw*₃ on chromosome 5 to the right of *bt*₁ in the long arm and *lw*₄ on chromosome 4, 13 units to the right of *su*₁ and 33 units to the left of *gl*₃. No further linkage data for these mutants are available at present.

Chlorophyll-1: This mutant was first placed to chromosome 3 by means of linkage with *T3-9c*. Allele tests have turned up two Group 1 alleles, *w*₇₇₁₆ and *w*_{pioneer}.

Linkage data from Table 15 indicate that *cl*₁ is located between *ra*₂ and *lg*₂ and to the left of the break point of *T3-9c* which LONGLEY (1958) placed at 3L.09. Table 16 (first and second crosses) summarizes all available linked information with this translocation and indicates 4.6 percent crossing between *cl*₁ and the break point in chromosome 3. Table 16 (third–fifth crosses) gives the results of two point tests involving *ra*₂ and *lg*₂. Since ANDERSON and RANDOLPH (1945) indicate *lg*₂ is well out in the long arm of chromosome 3 and since *cl*₁ is very close to the break point of *T3-9c* at 3L.09, it is very likely that these three genes lie in the order given. The results of linkage studies with *cl*₁ indicate the following map: *ra*₂—11.9—*cl*₁—4.6—*T3-9c*—22.7—*lg*₂. Because of the small numbers of plants tested in the three point test with *T3-9c*, the placement of *cl*₁ with respect to this translocation is still rather tenuous. The *cl*₁ locus must be very close to the centromere in chromosome 3.

*White*₇₇₁₈: This is a new Group 2 mutant that has not been described before. It first appeared in stocks derived from irradiated seeds planted at the California Institute of Technology. One Group 2 allele, *w*₈₆₂₄, has been found.

TABLE 15

Summary of three point data from testcrosses involving *cl*₁

Genotype of F ₁	Parental combinations		Recombinations						Percent recombinations		
			Region 1		Region 2		Region 1 and 2		Totals	Region 1	Region 2
$\frac{ra_2 + +}{+ cl_1 T3-9c}$	31	45	5	9	0	0	0	0	90	15.6	0
$\frac{+ + lg_2}{cl_1 T3-9c +}$	34	36	2	3	4	4	0	0	83	6.0	9.6
$\frac{ra_2 + +}{+ cl_1 lg_2}$	61	60	8	9	39	23	2	2	204	10.3	32.4

TABLE 16

Summary of two point data from testcrosses involving cl_1

Genotype of F_1	Parental combinations		Recombinations		Totals	Total recombinations	Percent recombinations
$\frac{+ T3-9c}{cl_1 +}$	189	190	11	8	398	19	4.8
$\frac{+ +}{cl_1 T3-9c}$	62	61	3	2	128	5	3.9
					526	24	4.6
$\frac{ra_2 +}{+ cl_1}$	213	248	32	30	523	62	11.9
$\frac{+ lg_2}{cl_1 +}$	79	98	26	26	229	52	22.7
$\frac{+ +}{cl_1 lg_2}$	68	70	25	41	204	66	32.4
					433	118	27.3

Indications that this locus was linked with waxy in the presence of $T3-9c$ were obtained on F_2 ears, suggesting that this gene was on chromosome 3. Testcross data (Table 17, first cross) with this translocation indicate that w_{7748} is 20.3 units from the break point in chromosome 3.

Heterozygous w_{7748} plants when pollinated by $B-3b$ produced kernels that gave rise to albino seedlings. Thus, this gene must be located in the distal nine tenths of the long arm of chromosome 3, the portion indicated by ROMAN and ULSTRUP (1951) that is attached to the B centromere in this translocation. Since LONGLEY (1958) has placed the break point of $T3-9c$ at 3L.09 (approximately the same as that of $B-3a$) and since 20.3 percent recombination is observed with this translocation, w_{7748} must be distal to $T3-9c$.

TABLE 17

Summary of two point data from testcrosses involving w_{7748}

Genotype of F_1	Parental combinations		Recombinations		Totals	Total recombinations	Percent recombinations
$\frac{T3-9c +}{+ w_{7748}}$	67	82	22	16	187	38	20.3
$\frac{+ lg_2}{w_{7748} +}$	73	91	10	12	186	22	11.8
$\frac{ra_2 +}{+ w_{7748}}$	63	80	28	41	212	69	32.5
$\frac{is_{24} +}{+ w_{7748}}$	65	63	13	19	160	32	20.0

Table 17 (second-fourth crosses) gives the results of two point tests with lg_2 , ra_2 and ts_4 . Using the data from these linkage studies the following map can be constructed: $ra_2-12.5-ts_4-20.0-w_{7748}-11.8-lg_2$.

Pastel₈₅₄₉: This Group 3 mutant was found in the progeny of a seed grown at the California Institute of Technology that was exposed to a gamma ray dose of 40,000r units. This mutant and its allele, white mutable, which have been described by ROBERTSON and ANDERSON (1961) are allelic to γ_1 . Since all linkage tests have given results consistent with the known linkage relationships of the γ_1 locus on chromosome 6 no linkage data for these pastel alleles will be given here.

Albescent: This Group 3 mutant was first described by PHIPPS (1929) and placed on chromosome 2 by PERRY and SPRAGUE (1936). In his linkage map, RHOADES (1954) has placed it two units proximal to ws_3 and seven units distal to lg_1 on chromosome 2. No further linkage data on this mutant are available at the present time.

SUMMARY

Available linkage data for 13 pigment deficient mutants of maize are summarized. The results of these studies are tabulated below (Linkage information given is based on data from this report unless indicated otherwise):

vp_2	chromosome 5:	$a_2-3.3-vp_2-0.9-ps-3.3-bm_1$
vp_5	chromosome 1:	$sr-0.8-vp_5-23.4-ts_2$
vp_9	chromosome 7:	$vp_9-10.9-gl_1-17.9-T7-9a$
ps	chromosome 5:	See vp_2
w_3	chromosome 2:	$v_1-23.9-w_3-33.8-Ch$
lw_1	chromosome 1:	$Kn-(1.1)-lw_1-23.8-bm_2$
lw_2	chromosome 5:	$pr-2.2-lw_2-gl_3-18.3-T5-9a$
cl_1	chromosome 3:	$ra_2-11.9-cl_1-27.3-lg_2$
w_{7748}	chromosome 3:	$ts_4-20.0-w_{7748}-11.8-lg_2$
pas_{8549}	chromosome 6:	Allele to γ_1
al	chromosome 2:	$ws_3-4-al-7-lg_1$ (RHOADES 1954)
lw_3	chromosome 5:	Duplicate factor with lw_4 . To right of bt , in long arm (TULPULE 1954)
lw_4	chromosome 4:	Duplicate factor with lw_3 . $su_1-13-lw_4-33-gl_3$ (TULPULE 1954)

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