produce a silk although not necessarily. This is analogous to the commonest effect produced in Pt ears.

When both tassel and ear are affected, there is general agreement between the severity of the effect on both. And when the phenotype of the ear is "inhibited" as is the case with many Pt/Pt plants, then the tassel also shows some degree of inhibition, i.e. restricted spikelet development.

The genetic background in which the effects of Pt are extended to the tassel has not been characterized although it is probably not complex. The best source was the linkage tester for Chr. 8 carrying i, v_{16}, ms_8 which was obtained from the Coop (50-55).

Oliver E. Nelson, Jr.

3. Double mutants in the chromosomal vicinity of a mutable locus.

The mutable allele a^{Pm} produces a high rate of mutation at the A_1 locus (News Letter 30: 111). This allele mutates both somatically and germinally so that deep, pale, light pale and colorless levels of anthocyanin pigmentation are expressed in the aleurone tissue. The alleles produced by germinal mutation vary in stability from stable (a^{P5}, no mutants in 30,000 tested gametes) to moderate stability (a^{b1}, 1 mutant per 13,000 gametes) to moderate mutability (A^1, 1 mutant per 4,000 gametes) to instability as marked as that in the parent allele.

Four of these new deep alleles, six new pale alleles, and four of the colorless alleles were examined for rates of mutation to stable alleles giving different levels of aleurone pigmentation. The results are given in Table 1. Two cases of coincident mutations at two loci occurred among the 158 mutants. In these cases the deep (A^4) and the pale (a^{a1}) alleles mutated to alleles expressing the colorless level while the adjacent dominant shrunken-2 allele assumed the recessive form. These mutants will be designated $\frac{1}{a\ sh_2}$ and $\frac{2}{a\ sh_2}$ respectively. One of these mutants, $\frac{1}{a\ sh_2}$, has been tested further and has been shown to behave in a manner similar to that of the a-X1 mutant of Stadler and Roman.

Although there is no visible indication of pollen abnormality in plants heterozygous for the double mutant ($a\ sh_2/\frac{1}{a\ sh_2}$), the transmission of the microgametophyte carrying $\frac{1}{a\ sh_2}$ is reduced. Table 2 shows a good deal of variation in the degree of transmission among the different cultures. The average per cent of normal transmission for these five cultures is 44.

Table 1. Rates of mutation in alleles derived from $\underline{a}^{Pm}$.

Allele tested	Gametes tested	Stable Mutants	Rate of Mutation
a^{P5}	30,538	0	-
a^{5}	29,527	0	-
a^{P1}	16,152	0	-
A^{2}	4,007	0	-
a^{P2}	2,096	0	-
a^{4}	24,010	1	1 per 24,010
a^{P3}	42,392	2	1 per 21,196
a^{b1}	26,540	2	1 per 13,270
A^{1}	36,379	8	1 per 4,547
a^{b2}	15,103	5	1 per 3,051
A^{4}	172,146	77	1 per 2,238
A^{R}	14,470	7	1 per 2,064
a^{P4}	22,525	15	1 per 1,501
a^{a1}	36,762	41	1 per 897

Table 2. Male transmission of $\frac{1}{\underline{a}\ \underline{sh_2}}$ in competition with $\underline{a}^{b1}\ \underline{Sh_2}$ as measured by percentage of normal transmission.

Cross	Total kernels	Sh_2	sh_2	% of sh_2	% of normal transmission
$A^{d}sh_2/A^{d}sh_2$ x $a^{b1}Sh_2/\frac{1}{a\ sh_2}$	1722	1343	379	22	28
$A\ sh_2/A\ sh_2$ x $a^{b1}Sh_2/\frac{1}{a\ sh_2}$	911	679	232	26	34
$A\ sh_2/A\ sh_2$ x $a^{b1}Sh_2/\frac{1}{a\ sh_2}$	1128	762	366	32	48
$a\ sh_2/a\ sh_2$ x $a^{b1}Sh_2/\frac{1}{a\ sh_2}$	3048	2667	381	12	14
$a\ sh_2/a\ sh_2$ x $a^{b1}Sh_2/\frac{1}{a\ sh_2}$	1998	1634	364	18	23

Transmission of $\frac{1}{\underline{a}\ \underline{sh_2}}$ through the megagametophyte was found to be normal. Data on this point are available by making use of the inability of the double mutant to dot in the presence of the gene Dt. Five ears resulting from the cross $\frac{1}{\underline{a}\ \underline{sh_2}}/\underline{a}\ \underline{sh_2}dt/dt \times \underline{a}^{b_1}\underline{Sh_2}/\underline{a}^{b_1}\underline{Sh_2}Dt/Dt$ produced 1084 kernels, 402 of which had dots. Independent data from closely related material show that only 70% of the endosperms of the constitution $\underline{a}\ \underline{sh_2}/\underline{a}\ \underline{sh_2}/\underline{a}^{b_1}\underline{Sh_2}$, Dt/dt/dt actually dot. If the 402 dotted kernels make up 70% of the $\underline{a}\ \underline{sh_2}$ gametes, then the total number of kernels carrying $\underline{a}\ \underline{sh_2}$ is 574. Hence the $\underline{a}\ \underline{sh_2}/\underline{a}\ \underline{sh_2}/\underline{a}^{b_1}\underline{Sh_2}$, Dt/dt/dt endosperms make up 52.8% of the total, and the $\frac{1}{\underline{a}\ \underline{sh_2}}$ carrying gametes make up 47.2% of the total functional gametes.

The similarities between $\frac{1}{\underline{a}\ \underline{sh_2}}$ and a-X1 may be summarized as follows:

1) Both have reduced male transmission.

2) Different stocks give rise to wide differences in degree of reduction of male transmission.

3) Both have normal egg transmission.

4) Both are unable to dot in the presence of Dt.

5) In both cases homozygotes are lethal.

6) The A_1 and Sh_2 loci are included in both cases.

The question arises whether these mutations are actually deficiencies as in the case of a-X1 or whether they are regions inactivated by the adjacent mutable locus. No conclusive evidence on this point is available, but small scale tests have failed to reveal reverse mutations at either the A_1 or Sh_2 locus.

Double mutants of this type are expected under the following hypothetical model for the structure of a^{Pm}. In this scheme the components of a^{Pm} are: β, the deep pigment producing factor, α, the unit responsible for pale pigmentation, P^b, the dominant brown pericarp factor, and M, the mutability factor. These components are arranged in the following order:

----Centromere-----β-----M-----α-----P^b-----Sh_2-----.

It is assumed that the mutability factor involved here, like others more thoroughly studied, is capable of inhibition of adjacent loci, and that this capacity to inhibit may spread along the chromosome in either direction. Further it is assumed that when two genes are inhibited in

this manner, all loci between them on the chromosome will also be inactivated.

The possible effects that various types of inhibition by M might produce are:

Inactivation of	Mutant Produced	Frequency of this Mutation
M	Stable, pale, dominant brown pericarp, Sh_2	Frequent
β M	Stable, pale, dominant brown pericarp, Sh_2	
M α	Stable, deep, dominant brown pericarp, Sh_2	No occurrence. (In very similar material, M. G. Nuffer has found this type of mutant which, however, is somewhat mutable.)
M α P^b	Stable, deep red pericarp, Sh_2	Nine cases
M α P^b Sh_2	Stable, deep, red pericarp, sh_2	Several deep sh_2 kernels arose in crosses of a^{pm} sh_2/a sh_2 x a sh_2/a sh_2 but were discarded as probable contaminations. (No analysis of pericarp constitution)
β M α	Stable, colorless, dominant brown pericarp, Sh_2	Seven cases
β M α P^b	Stable, colorless, recessive brown pericarp, Sh_2	Common
β M α P^b Sh_2	Stable, colorless, recessive brown pericarp, sh_2	Two cases (Not yet analyzed for pericarp constitution.)

Critical types of mutation which could not be explained by this hypothesis are:

Mutant Type	Requires Inactivation of	Requires Intermediate Components Unaffected
Stable, pale, recessive brown pericarp, Sh_2	β M - P^b	α
Stable, deep, dominant brown pericarp, sh_2	M α - Sh_2	P^b
Stable, pale, dominant brown pericarp, sh_2	β M - - Sh_2	α P^b
Stable, pale, recessive brown pericarp, sh_2	β M - P^b Sh_2	α
Stable, colorless, dominant brown pericarp, sh_2	β M α - sh_2	P^b

None of these critical types have been found as yet, but an extensive program for their detection is underway.

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1. Competitive growth of pollen tubes in maize.

In this preliminary investigation the possibility was considered whether there is a relation between combining ability and pollen tube growth.

The experimental procedure was to mix equal quantities of pollen of yellow and white seeded inbreds obtained from 15-20 plants in each case and to use this mixture to pollinate different white seeded inbreds and the white seeded variety Potchefstroom Pearl. The sources of pollen were the well known American inbreds K 64, 33-16 and Hy and the South African inbred A 413, all known for their good combining ability. In addition five local yellow inbreds and a white inbred P 697 all of unknown combining ability, and the white inbreds F60 and E58 of known weak combining ability were used as pollen parents. The maternal parents were the inbreds K64, 33-16, E58 and F60 all of known combining ability and 20 other white inbreds of unknown combining ability. Pollen mixtures of the good white combiner K64 with different yellow