

<u>5L</u>	<u>6L</u>	<u>7L</u>	<u>8L</u>	<u>9S</u>	<u>9L</u>	<u>10S</u>	<u>10L</u>
1 spl lvs	2 w	1 wl	1 wl	4 w	1 v	1 wl	1 l
	3 wl	1 g	3 v	1 sh	1 d	2 zb	1 v
	1 v	1 gl	1 nec	1 zb	1 ad	1 sr	3 pg
	1 nec	1 nl	1 de			2 ad	1 ad
		1 et					1 nec
		2 de					
		2 mn					

We welcome interest by fellow workers willing to determine linkage and allelism of mutants already placed to chromosome arm and would be pleased to send seed of all the mutants of a particular arm to those interested. Mutants on the following arms have already been taken: 3L, 7L, 8L, 9S, 9L and 10L.

Since we are routinely crossing large numbers of mutants by the full set of A-B translocations, we are willing to include in our plantings a limited number of mutants from fellow workers who have something that they feel is urgent. We prefer  $F_1$  seed. Send  $20^+$  kernels for each by May 1.

M. G. Neuffer  
J. B. Beckett

## 2. Absence of auxotrophic mutants in corn and other eukaryotes.

The failure to obtain obligate auxotrophic mutants in corn and other eukaryotes has been a puzzle, especially in view of the remarkable success with fungi and bacteria. Numerous attempts by the author and others to grow various types of lethal and sub-lethal mutants on supplemental media have been mostly unsuccessful. Only a few mutants have been found in higher plants (Gavazzi, *et al.*, MNL 47:114-121; Nelson and Burr, 1973, Annual Review of Plant Physiology 24:493-518; Li and Redei, 1969, Biochemical Genetics 3:163-170).

One possible explanation may lie in intercellular transfer of gene products. It is possible that the mutants that are commonly observed and studied are for genes whose product is not transferable (nondiffusible or cell limited or unstable). Failure of transfer is suggested by the fact that chimeras for most of the known recessive mutants in corn have distinct borders. Distinct borders are generally found for chimeras resulting from chromosome loss, from reversions

arising through the action of controlling elements and from spontaneous mutations.

If transfer is an important factor, what would be the phenotypic expression of various types of mutants with and without effective inter-cellular transfer of gene product? A series of predictions can be framed for the alternatives of transferable vs. nontransferable product and universally vs. stage-specific vital functions, such as chloroplast assembly or chlorophyll synthesis.

If one treats mature pollen with a mutagenic agent and produces in the pro-embryo nucleus a recessive mutant for a gene controlling a universally vital function involving a nontransferable product, one may expect the following consequences. The mutant will potentially be a cell lethal but will survive through the gametophyte generation because of the covering of the sperm by the tube nucleus and will form a viable zygote. The lethality of the mutant will not be tested until sporogenesis when, for the first time, cells will arise with only a mutant allele in the nucleus. These will lack the vital function and will abort. The phenotype will therefore be a normal appearing  $F_1$  plant with 50% aborted pollen and ovules (semisterile pollen and ear). The mutant will not be transmitted to the next generation.

A mutant that controls a vital function for which the product can be transferred will survive through the  $F_1$  as above, but the gametes produced will have the advantage of gene product from normal diploid cells of the supporting tissue. Thus, the gametes of the  $F_1$  plant may survive to achieve fertilization. (This may or may not be true for the microspores, since the  $\sigma^1$  gamete does go through a short period of independent existence.) Assuming both  $\sigma^1$  and  $\text{♀}$  gametes succeed, the selfed  $F_1$  will produce 1/4th homozygous mutant kernels. The mutant embryos may not be lethal at first, however, because the endosperm may carry stored gene product supplied by the ear parent. Lethality will occur when this endosperm supply is exhausted and the seedling must make its own product. The mutant would therefore be expressed in the  $F_2$  as a normal seedling that dies when the endosperm nutrients are exhausted (about the 3-leaf stage). The necrotic lethals appear to be of this kind. If the  $\sigma^1$  gamete does not survive, then only normal pollen grains will effect fertilization, and the  $F_2$

will include only normal plants. Half will be heterozygous, however. The mutant would escape detection unless special techniques such as differential transmission of linked markers are used to detect it.

A mutant that controls a stage-specific vital function such as chloroplast assembly and a product that is confined to the cell where it is produced should form viable mutant gametes because chloroplasts are not necessary for gametogenesis. The  $F_1$  selfed will produce 1/4th homozygous mutant embryos that will grow into chlorophyll-less seedlings which will survive only as long as the endosperm nutrients last. This type of mutant would be expressed in the  $F_2$  as white or yellow seedlings that die at endosperm depletion. The commonly occurring w, wl and l mutants would fit in this category.

A mutant that controls chlorophyll synthesis through a product that is transferable and stored may have  $F_2$  seedlings that initially are normal or nearly normal green (depending on the efficiency of transfer) if the ear parent supplies the gene product and it is present in the embryos and endosperm until depleted by the seedling. At depletion, the green seedling should deteriorate in one of two ways. If intact chlorophyll can continue to function without the product, the first 2-3 leaves will be normal green and subsequent leaves will be more white or yellow. The plant will live for some time on the photosynthesis of the first leaves, but eventually will die. If, however, the manufactured chlorophyll in the first leaves required continued gene product to function, at depletion the existing leaves will begin to fade to white or yellow or to discolor, and the seedling will die rather abruptly. Two alternative phenotypes would appear for the  $F_2$  of this type of mutant: (1) Initially green seedlings which produce newer leaves that are white or yellow. The mutants should survive to the 5th or 6th leaf stage. (2) Initially green seedlings which begin to fade rather abruptly to white or yellow and die. Mutants representing the first of these types have not been seen, while a number of the latter have been obtained as a result of treatment with EMS.

This sort of reasoning may lead to the recognition of a number of new classes of mutants.

M. G. Neuffer