I and En therefore appear to be related although they differ in activity. I inhibits the expression of the normal allele (Pg) whereas En inactivates or causes the removal of I. Whether the difference in activity of the two elements is a question of position or chemical composition can only be conjectured.

-- Peter A. Peterson

2. a_1-mutable.

It has now been confirmed that a^m found originally in pg^m stocks, has the same components (I and En) as the pg mutable system. Direction of mutation, pattern types, rate of appearance and types of stables, and the relationship of particular patterns to specific stables have been studied.

The direction of mutation: Many kernel patterns have been described. They vary from a very dense pigmentation type to small, infrequently spotted types. (The former result from early, the latter from late mutations.) Each of the distinct pattern types can give rise to other pattern types and each of these derivatives has been tested and found to be heritable. The events that lead to changed patterns occur at the a^m locus or result from the mutation of the autonomous controller of mutability to the independent type.

Rate of appearance and types of stables: The rate of occurrence of stables varies with the particular pattern. The higher rates are associated with the dense type mutable patterns. Although stables are phenotypically alike, some mutate in the presence of En, others do not. Thus the response to En is a means of distinguishing among the "stables." Particular patterns give rise to a designated type of stable. This is relevant to the analysis of the genetic events that accompany changes in pattern phenotype.

Factor causing dense kernels: In the last newsletter, it was reported that in the presence of a factor "D", a specific fine pattern allele becomes dense (appears full colored), and in its absence, the pattern remains fine. It has now been confirmed that this factor is En. The stable derivatives from the dense phenotype may contain En but do not respond to it.

-- Peter A. Peterson

3. Some thoughts on the white-albino mutants.

For the past ten years I have been accumulating albino mutants. Most of these have white (or pale yellow) endosperm and chalky-white, albino seedlings when germinated in the light. I have called these mutants white-albino, although other terms have been used to describe them such as lemon-white, viviparous, (because of the tendency of some to germinate prematurely), white, etc.

The pleiotropic effects of these genes are of some interest and several explanations have been suggested for these effects. Biochemical studies which have been made here (see below) suggest that the basic block is in the carotenoid synthesis of these mutants. The lack of chlorophyll might be due to a lack of phytol which most likely is synthesized via the carotenoid pathway. However, J. H. C. Smith (Stanford) and I. C. Anderson (Iowa State) have shown that some of these mutants synthesize chlorophyllide in the dark and also have sufficient phytol available to form chlorophyll, which suggests that the lack of chlorophyll in the light-grown seedlings might be due to a secondary factor such as the photodestruction of chlorophyll in the presence of oxygen when carotenoids are absent (see below).

The simultaneous involvement of pigment synthesis in the endosperm and seedling of these mutants is most easily explained by assuming that the mutated gene blocks carotenoid synthesis wherever it occurs. However, mutants are known where carotenoid synthesis of only one or the other of these tissues is involved, such as y-1, where only the endosperm and not the seedling is affected. The reciprocal class of mutants, with yellow seeds and albino seedlings, is also found in corn. To further complicate the picture we have pastel (pale-green) mutants which have the white endosperm but produce pale-green instead of albino seedlings. A summary of the different classes of mutants with defective pigment production in endosperm and seedling which we have been studying is given in table 1. Some of the mutants which do not have the typical white-albino phenotype nevertheless have been found to be allelic to them. For example, there has been found an allele for a white-albino mutant in which the endosperm is yellow and an allele to another in which the seedling is pale-green. An allele of this latter type has been found for y-1. A summary of the phenotypes of known alleles is given in table 2.

Table I. A summary of the different classes of pigment deficient mutants that have been studied at Iowa State.

Class of mutant	Phenotype of Endosperm	Phenotype of Plant
1	White	Albino
2	White	Green
3	White	Pale Green (pastel)
4	Yellow	Albino

Table II. A summary of alleles found for the white-albino mutants.

Genes involved in allelic series (included between lines)	Mutant classes of the alleles (see Table I.)
w-3	1
pas-8686	3
y-1	2
pas-8549	3
cl-1	1
(pas)-pioneer	2 or 3*
**vp-9	1
**pas-4889	3
**w-8657	4

* Actual phenotype of (pastel)-pioneer is somewhat uncertain at the present time. These two mutants are definitely alleles on the basis of endosperm phenotype. However, the homozygous (pastel)-pioneer plants will have to be grown in the field next year before a definite plant phenotype can be established.

** This last summer there was obtained one segregating F_1 ear between vp-9 and pastel-4889 and one segregating F_1 ear between pastel-4889 and w-8657. These results will have to be confirmed next year and the F_1 between vp-9 and w-8657 obtained before this series can be established as certain.

The information summarized in tables 1 & 2 would suggest that the endosperm and seedling phenotypes can be modified independently. Dr. Everett has shown a similar independent modification of seedling and endosperm by the use of suppressor genes on the cl -1 and other loci.

In order to explain the independent modification of endosperm and seedling in these mutants it can be assumed that we are dealing with two closely linked loci, one concerned with pigment production in the endosperm and the other with pigment production in the seedling (plant). However, the fact that many (20 or more counting alleles) of these white-albino mutants have occurred spontaneously or were induced by irradiation, would suggest that this was not so. It is extremely unlikely that the two closely linked loci would mutate simultaneously in all ofthese instances. Another possibility is that we are dealing with complex gene loci that consist of at least two parts. One portion is involved in the synthesis of pigment in the endosperm and the other in the seedling. Allele tests would suggest that the different portions of the complex gene could mutate simultaneously or independently and also, that the plant portion is capable of mutating in two ways: 1) complete inactivation resulting in no pigment formation or 2) partial inactivation, resulting in pale-green plants. The latter could be accomplished by a change in a portion of the plant part of the complex (suggesting a further subdivision of the locus) or by a more subtle rearrangement of the plant unit as a whole. See figure 1 for a summary of the possible alleles.

There are several lines of evidence that can be used to determine if the white-albino mutants are indeed complex loci. Two of these are circumstantial and have been mentioned above. One is by finding suppressor genes (such as Dr. Everett and colleagues have been doing) that suppress the mutant phenotype in one or the other of the tissues. The other line of evidence that has been mentioned is the finding of allelic series of genes with different endosperm and seedling phenotypes. A third line of evidence would be to separate the two portions of the complex by crossing over. We are concentrating on the latter two lines of evidence here.

Such complex loci could be similar in structure to the R locus as revealed by Stadler and Emmerling, where it has been possible to demonstrate seed and plant sub-units. However, the white-albinos differ in one important regard from the R-locus alleles in that mutation can proceed from yellow endosperm-green plant (equivalent to R^r) to white endosperm-albino plant (equivalent to r^g) in one step.

The presence of a seed and plant unit at the R locus and the possibility of a similar compound structure for these many white-albino mutants suggest that a compound structure might be characteristic of some of the other genes that have obvious phenotypic effects in seed and plant, such as A-1 (compound structure has already been demonstrated by Laughnan but not divided along the lines indicated above) and A-2.

We would like to solicit the help of other corn workers in finding alleles of the types described above to these white-albino mutants.

We would particularly be interested in finding white endosperm mutants with green plants (other than y-1), white endosperm pale-green mutants, and yellow endosperm chalky-white albinos. Additional white-albino mutants would also be welcome.

-- Donald S. Robertson