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).