be possible, by studying strains from asci with both normal and defective spores, to show that the cross of *pyr-3a* to *pyr-3b* yields strains which are not phenotypically wild but from which both parent mutants can be recovered. If so, investigation in this manner of other *pyr* mutants which are phenotypically similar to *pyr-3a* and *-3d* may prove to be of interest in showing how many types there are with respect to the ability to produce, in intercrosses, strains of this kind which are phenotypically wild.

* This work was supported in part by funds from the Rockefeller Foundation and by funds from the Atomic Energy Commission administered through contract with the Office of Naval Research, U. S. Navy, Contract N6-onr 244, Task Order V.

[1] Horowitz, N. H., Bonner, D. M., and Houlahan, M. B., *J. Biol. Chem.*, **159**, 145 (1945).

[2] Mitchell, H. K., and Houlahan, M. B., *Fed. Proc.*, **6**, 506 (1947).

[3] Mitchell, M. B., and Mitchell, H. K., PROC. NATL. ACAD. SCI., **38**, 205 (1952).

[4] McClintock, B., *Am. J. Bot.*, **32**, 671 (1945).

[5] Houlahan, M. B., Beadle, G. W., and Calhoun, H. G., *Genetics*, **34**, 493 (1949).

[6] Kölmark, G., and Westergaard, M., *Hereditas*, **25**, 490 (1949).

[7] Srb, A. M., and Horowitz, N. H., *J. Biol. Chem.*, **154**, 129 (1944).

[8] Singleton, J. R., unpublished Ph.D. Dissertation, California Institute of Technology (1948).

[9] Westergaard, M., and Mitchell, H. K., *Am. J. Bot.*, **34**, 573 (1947).

[10] Dodge, B. O., *J. Agr. Res.*, **35**, 289 (1927).

[11] Lawrence, W. J. C., *J. Genetics*, **24**, 257 (1931).

THE GENOTYPE OF THE ENDOSPERM AND EMBRYO AS IT INFLUENCES VIVIPARY IN MAIZE

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Communicated by G. W. Beadle, May 13, 1952

The development of the maize seed is dependent on the orderly unfolding of events in which each component of the developing caryopsis has a particular role to play. The ultimate control of these events must depend upon numerous genes, which if altered will interfere with normal development. Many mutants of this type have been described, ranging from those which produce relatively slight alteration in the caryopsis to those which prevent practically all development. Among those producing relatively slight changes are mutants which give rise to premature germination. The seeds of these mutants develop normally until late in ontogeny. During the early dough stage the plumule begins to elongate, and the seeds germinate while still attached to the ear. Such mutants have been called viviparous.

The viviparous gene may induce changes in one or more of the various tissues of the developing caryopsis. Since viviparous seeds segregate on the ear, any changes induced in maternal tissue cannot determine whether a seed will be viviparous. Changes responsible for vivipary must be produced in tissues resulting from the union of male and female gametes—the embryo and/or the endosperm. There are three possible ways that the viviparous gene can promote premature germination: (1) by inducing in the embryo changes which allow it to germinate under conditions normally not conducive to germination; (2) by inducing in the endosperm changes which alter the normal germination inhibiting mechanism; and (3) by inducing changes in both the endosperm and embryo which interact to overcome the normal germination inhibition. In order to determine which of these three possibilities actually is operative within the mutant seed, it is first necessary to isolate the effect produced by the viviparous genotype of the endosperm from that of the embryo.

Seeds resulting from hetero-fertilization frequently have embryos and endosperms of different genetic constitution. Such seeds are produced when the egg and polar nuclei of the egg sac fuse with sperm nuclei of different

TABLE 1

	GENOTYPE OF THE ENDOSPERM	GENOTYPE OF THE EMBRYO
Class A	*Vp Vp vp vp*	*vp*
Class B	*vp vp*	*Vp Vp vp*

pollen grains. Sprague[1] observed a few such seeds on an ear segregating for one of the viviparous mutants. He found that only the seeds with genetically viviparous embryos germinated. This would indicate that in this mutant, at least, the gene effects changes in the embryo which induce germination.

The use of A-B translocations makes possible the systematic production of embryos and endosperms of differing genetic constitution. In some strains of maize there may be present, besides the basic set of ten A-type chromosomes, one or more supernumerary, genetically inert, chromosomes which have been designated as B-type. Roman[2] has described the behavior of A-B interchanges in the production of the male gametes. In the division of the generative nucleus of the developing pollen grain the B centromeres usually fail to separate, giving one sperm both B centromeres and the other none. Where a portion of an A-type chromosome is attached to the B centromere, this non-disjunction produces within a given pollen grain two sperm of different genetic constitution. One sperm has the portion of the chromosome attached to the B centromere present in duplicate (hyperploid sperm), whereas the other is deficient for this region (deficient sperm). If the viviparous gene is located in the trans-

located section of the A chromosome, two classes of seeds are obtained from pollination of viviparous by an A-B interchange. These two classes are shown in table 1. Class A seeds are the result of the hyperploid sperm fusing with the polar fusion nucleus, and the deficient sperm fusing with the egg nucleus; whereas Class B seeds are the result of the reciprocal fusion. In both classes the endosperm and embryo differ with reference to their viviparous genotype.

Although these classes are theoretically possible, they are not readily recognized by the expression of vivipary alone. If one of these classes of seeds were to germinate prematurely, there is no way to determine *a priori* to which class it belongs. However, it is possible to distinguish these classes in some viviparous mutants which interrupt the normal carotenoid and chlorophyll development. Seeds of these mutants have white or pale yellow endosperms and germinate prematurely, producing albino seedlings. By using such a mutant in combination with the A-B interchanges, it is possible to recognize the two types of seeds shown in table 1. Class A seeds should have yellow endosperms because they carry the normal alleles of vivipary and produce white seedlings, since only the mutant allele is present in the embryo. The Class B seeds would be recognizable by white endosperms and green seedlings.[6]

Plants heterozygous for viviparous-5, a mutant which inhibits carotenoid and chorophyll development as well as inducing premature germination, was pollinated by pollen of plants carrying A-B interchange TB-1b. The resulting ears were found to be segregating viviparous and white seeds. A classification of endosprem and seedling color revealed three types of seeds: (1) yellow endosprem with albino seedlings, (2) white endosperm with green seedlings, and (3) yellow endosperm with green seedlings. Since viviparous-5 affects carotenoid formation, it is possible to recognize the first type of seeds as those belonging to Class A. The second type of seeds are those belonging to Class B. The third type results from the fertilization of ovules carrying the normal alleles of viviparous-5 and are, therefore, not important in this consideration. Thus, the two classes of seeds shown in table 1 are recognizable. Only seeds of Class A were found to be viviparous, whereas those of Class B were dormant. As is expected, all seeds of Class B grown to maturity and self-pollinated have segregated for viviparous-5.

By using TB-1b in combination with viviparous-5, it has been possible to distinguish between the role of the endosperm and that of the embryo in the production of premature germinating seeds. The data suggest that the endosperm does not play an active role in vivipary, since seeds germinate prematurely if they have a normal or mutant endosperm as long as the embryo is genetically viviparous. Thus, it would appear that vivipary is determined by the genotype of the embryo and is independent of the

genotype of the endosperm. The evidence from hetero-fertilization supports this conclusion.

The nature of the mechanism responsible for the dormancy of the developing embryo is not known. Work of La Rue,[4, 5] Haagen-Smit *et al.*,[6] and others has shown that normal corn embryos are capable of germination soon after pollination, if removed from the developing caryopsis and cultured. Thus, the developmental dormancy is not entirely dependent upon the embryo but is influenced by some environmental factor within the developing seed. The genetic evidence and that from embryo culture experiments suggest that viviparous seeds germinate prematurely because their embryos are capable of germination within the caryopsis in an environment which will not permit the germination of normal embryos.

* This paper is based on work done at the experimental farm of the California Institute of Technology. I am indebted to Dr. E. G. Anderson for his advice and encouragement.

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[1] Sprague, G. F., *Am. Soc. Agronomy*, **28**, 472–478 (1936).

[2] Roman, H., *Genetics*, **32**, 391–409 (1947).

[3] The validity of these conclusions is based on the observation that the inhibition of carotenoid and chlorophyll development by the mutant genes is not dependent on its first inducing vivipary.

[4] La Rue, C. D., *Bull. Torr. Bot. Club*, **63**, 365–382 (1936).

[5] La Rue, C. D., *Science*, **115**, 315 (1952).

[6] Haagen-Smit, A. J., Sui, R., and Wilson, G., *Science*, **101**, 234 (1945).

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*SUBSTRATE STABILIZATION OF ENZYME-FORMING CAPACITY DURING THE SEGREGATION OF A HETEROZYGOTE**

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Communicated by M. M. Rhoades, May 23, 1952

Introduction.—In the course of investigating the inheritance of the capacity to synthesize "galactozymase" Winge and Roberts[1] discovered a "slow" adapting phenotype. These authors demonstrated that the character was determined by a single recessive allele (g_s) which segregates normally in crosses to the wild type bearing the dominant (G).

Analysis by Spiegelman, Sussman and Pinska[2] of g_s-genotype cultures revealed the existence of heritable differences in phenotype among cells carrying this allele. This heterogeneity is easily and simply exhibited by plating glucose-grown g_s-cells on purified galactose agar test plates upon