

second mutant gene. However, fewer backcrosses would be required than if an unrelated non-recurrent parent were used with conventional backcross procedures.

Apparently EMS can be used to produce cytoplasmic mutants in plants (Dulieu, Mutation Res. 4:177-189, 1967) and may be useful to produce cytoplasmic sterility in maize by the above outlined procedures; probably more important, cytoplasmic sterility may be produced in other species with some modification of these procedures.

Robert W. Briggs

2. Modification of the efficiency and effectiveness of ethyl methanesulfonate treatments in maize.

The action of ethyl methanesulfonate (EMS) on seeds was investigated by altering post-treatment conditions so as to modify the genetic effects and physiological damage produced. Genetic effects were measured by the frequency of yellow green ( $yg_2$ ) sectors in the leaves of  $Yg_2/Yg_2$  seedlings; physiological damage by reduction in plant height. If seeds are dried immediately after treating with 0.01 M EMS (10 h, 25° C), the height of seedlings will be reduced significantly. If, however, the seeds are soaked for 4 days (at 3° C) after EMS treatment, then dried, the physiological damage is minimized and equals the control value. This post-treatment condition also reduces mutation rate, but it maximizes the treatment efficiency (ratio of  $yg_2$  frequency to plant height reduction). Post-soaking apparently removes EMS and its hydrolysis products which are particularly harmful when the seeds are dried. However, post-soaking EMS-treated seeds before drying reduced the effectiveness of the treatment, as measured by the ratio of  $yg_2$  frequency to dose of mutagen. This is probably because unhydrolyzed (active) EMS was removed from the seeds.

Robert W. Briggs

3. Esterase isozymes: new loci.

The  $E_1$  through  $E_4$  esterases were described by Drew Schwartz and his students at Indiana. The present note briefly describes several new anodal esterase loci and a single transaminase locus found during an ongoing classification of enzyme polymorphisms in flowering plants carried out at the University of Hawaii.

Re<sub>4</sub>. This locus interacts with 'prime' alleles at the E<sub>4</sub> esterase locus to cause cessation of enzyme production. Two alleles were found: Re<sub>4</sub>, which is functional with respect to interacting with the prime E<sub>4</sub> locus, and Re<sub>4</sub><sup>o</sup>, which is nonfunctional and does not interact with the E<sub>4</sub> locus.

Oe<sub>4</sub>. The Oe<sub>4</sub> site represents the interacting, or regulatory portion of the E<sub>4</sub> locus. Linkage between the Oe<sub>4</sub> and the E<sub>4</sub> sites has not been broken and, at present, they must be considered part of the same locus. Two 'alleles' of the Oe<sub>4</sub> 'locus' were found. Oe<sub>4</sub> represents the regulatory portion of a prime allele (designated as Oe<sub>4</sub>E<sub>4</sub><sup>n</sup>, where n = one of the electrophoretic variants of the E<sub>4</sub> esterase isozymes). Plants containing the prime allele and the functional Re<sub>4</sub> 'regulator' allele do not produce E<sub>4</sub> esterase isozymes. The other 'allele', Oe<sub>4</sub><sup>c</sup>, represents the regulatory portion of a standard allele (designated as Oe<sub>4</sub><sup>c</sup>E<sub>4</sub><sup>n</sup>, where n = one of the electrophoretic variants of the E<sub>4</sub> esterase isozymes). The standard allele is not affected by the Re<sub>4</sub> locus and is of the 'constitutive' type.

To summarize then, two independently segregating loci control the E<sub>4</sub> esterase isozymes. Prime E<sub>4</sub> alleles interact with the functional 'regulator' allele of the Re<sub>4</sub> locus resulting in cessation of enzyme production. Prime alleles, in the presence of the nonfunctional 'regulator' allele, and standard alleles produce functional E<sub>4</sub> esterase isozymes.

It should be pointed out that in addition to the null phenotype produced through the interaction of prime alleles and functional 'regulator' alleles, null phenotypes were also produced due to nonfunctional 'structural' portions of the E<sub>4</sub> locus (E<sub>4</sub><sup>null</sup>).

The genetic designations proposed for the E<sub>4</sub> esterases are not meant to imply that the prime and standard alleles represent operons. It has not been shown that the Oe<sub>4</sub> and the E<sub>4</sub> portions of this locus represent discrete cistrons due to the lack of crossover data. If one were to draw analogies, however, the Oe<sub>4</sub> portion of the locus would represent the operator gene of an operon and the E<sub>4</sub> portion of the locus would represent the structural gene of an operon.

E<sub>5</sub> Esterases. Two loci are involved in the control of the E<sub>5</sub> esterases. Two phenotypes were observed and each consisted of four

isozymes arranged in two pairs. Complex-I was assigned to inbreds containing the faster migrating pairs of isozymes while Complex-II was assigned to inbreds containing the slower migrating pairs of isozymes. Genetic analysis led to the postulating of two loci ( $\underline{E}_5$ -I and  $\underline{E}_5$ -II) which segregate independently. Each locus contained two alleles ( $\underline{E}_5$ -I<sup>A</sup>,  $\underline{E}_5$ -I<sup>a</sup> and  $\underline{E}_5$ -II<sup>A</sup>,  $\underline{E}_5$ -II<sup>a</sup>). Complex-II was found only in double recessive individuals ( $\underline{E}_5$ -I<sup>a</sup>/ $\underline{E}_5$ -I<sup>a</sup>;  $\underline{E}_5$ -II<sup>a</sup>/ $\underline{E}_5$ -II<sup>a</sup>). All other combinations resulted in Complex-I.

$\underline{E}_6$  Esterases. Two phenotypes were noted for the  $\underline{E}_6$  esterases. A single isozyme was either present or absent in inbreds. A simple monogenic control mechanism was indicated from the genetic analysis. Two alleles were postulated ( $\underline{E}_6^A$  and  $\underline{E}_6^{\text{null}}$ ). Individuals homozygous for the null allele showed no  $\underline{E}_6$  esterase isozyme. The heterozygote and homozygous  $\underline{E}_6^A$  individuals contained the isozyme.

$\underline{E}_7$  Esterases. Two phenotypes were noted for the  $\underline{E}_7$  esterases. A pair of closely associated isozymes was either present or absent in inbreds. Genetic analysis indicated that a single locus controlled the  $\underline{E}_7$  esterases and two alleles were proposed ( $\underline{E}_7^A$  and  $\underline{E}_7^{\text{null}}$ ). Individuals homozygous for the null allele lacked the  $\underline{E}_7$  esterase isozymes. The heterozygote and homozygous  $\underline{E}_7^A$  individuals contained the isozymes.

$\underline{E}_8$  Esterases. Two phenotypes were noted for the  $\underline{E}_8$  esterases. A single isozyme was either present or absent in inbreds. Genetic analysis indicated that the  $\underline{E}_8$  esterases were controlled by a single locus and two alleles were postulated ( $\underline{E}_8^A$  and  $\underline{E}_8^{\text{null}}$ ). Individuals homozygous for the null allele lacked the  $\underline{E}_8$  isozyme. The heterozygote and homozygous  $\underline{E}_8^A$  individuals contained the isozyme. This isozyme is inhibited by fluoride.

$\underline{E}_9$  Esterases. Three phenotypes were observed to be controlled by a single locus. Three alleles were postulated.  $\underline{E}_9^A$ , when homozygous, demonstrated a single isozyme which migrated faster than the isozyme demonstrated in  $\underline{E}_9^B$  homozygotes. The third allele,  $\underline{E}_9^{\text{null}}$ , lacked both of the isozymes when homozygous. Heterozygotes between  $\underline{E}_9^A$  and  $\underline{E}_9^B$  contained both isozymes. Heterozygotes between  $\underline{E}_9^A$  and  $\underline{E}_9^{\text{null}}$  contained the faster isozyme. Heterozygotes between  $\underline{E}_9^B$  and  $\underline{E}_9^{\text{null}}$  contained the slower isozyme. The two isozymes of the  $\underline{E}_9$  locus were activated by atropine.

E<sub>10</sub> Esterases. The E<sub>10</sub> esterases were confined to immature endosperm tissue. Two phenotypes were noted for the E<sub>10</sub> esterases. Individuals homozygous for E<sub>10</sub><sup>A</sup> demonstrated a single isozyme which migrated faster than the single isozyme found in E<sub>10</sub><sup>B</sup> homozygotes. The heterozygote E<sub>10</sub><sup>A</sup>/E<sub>10</sub><sup>A</sup>/E<sub>10</sub><sup>B</sup> demonstrated both isozymes; however, due to the dosage effect the faster isozyme was stronger than the slower isozyme. The heterozygote E<sub>10</sub><sup>A</sup>/E<sub>10</sub><sup>B</sup>/E<sub>10</sub><sup>B</sup> also demonstrated both isozymes, but in this case the slower isozyme was stronger than the faster isozyme. A single locus was postulated.

Ta<sub>1</sub> Transaminases. Two transaminase variants were found to be controlled by a single locus. The symbol Ta<sub>1</sub> was assigned to this locus and two alleles were noted (Ta<sub>1</sub><sup>A</sup> and Ta<sub>1</sub><sup>B</sup>). Ta<sub>1</sub><sup>A</sup> homozygotes showed the faster variant. Heterozygotes demonstrated three isozymes: the faster variant associated with Ta<sub>1</sub><sup>A</sup> homozygotes, the slower variant associated with Ta<sub>1</sub><sup>B</sup> homozygotes, and a third isozyme intermediate in migration between the two parental types. It was postulated that the functional transaminase isozyme is dimeric in structure and that the intermediate isozyme represents an allodimer or hybrid enzyme containing the two parental type subunits. The faster isozyme would then be composed of two A type subunits, the slower would be composed of two B type subunits, and the hybrid isozyme would contain one of each. The transaminase isozyme migrated towards the anode at pH 8.2.

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1. Studies on the transmission of the B type chromosomes in maize.

Chromosome morphology studies made by Longley and Kato (1965) and Kato (1964) on different Mexican maize varieties have shown that some tend to accumulate a high number of B type chromosomes. Other varieties present very low frequencies of this type of chromosome and still other populations have no B chromosomes.