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1. On the use of the TB-3a translocation to localize the lethal effects of the interaction of the mutant etched allele and M^{et}.

In M.G.C.N.L. 40:39-42, 1966, I reported on a system of zygotic lethality involving the interaction of the recessive etched allele of chromosome 3 and the previously unreported modifier, M^{et}. Individuals homozygous for the modifier, $M^{et}M^{et}$, and heterozygous for the etched locus produce no etched kernels as a result of selfing or testcrossing by standard etched testers. The elimination of etched individuals was demonstrated to be postzygotic in nature by genetic and histological tests. At that time we had in hand at least circumstantial evidence that the elimination of etched individuals was based on the existence of two or more "doses" of the modifier, M^{et}, in endosperm tissues and was totally independent of the modifier genotype of the embryo.

In order to verify that this is the actual situation, we turned to the use of the TB-3a translocation. Previous studies have indicated that the dominant allele of the etched locus (Et) overcomes the lethality conditioned by the interaction of et and the modifier M^{et}. It was reasoned that a TB-3a stock, lacking the mutant etched allele but homozygous for the modifier, could be established and used to vary the "doses" of et in the endosperm and embryo as a result of post-meiotic non-disjunction of the B-centromere (see Table 1.) Such a test should allow us to localize the lethal effect of this system of genic interaction in either the endosperm or embryo.

TB-3a tester stocks of the appropriate modifier genotype have been established and some crossing was done this past summer. The analysis of the data from these crosses is incomplete as of this writing because further field testing is necessary.

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2. On the nature of the interaction of M^{et} and the mutant etched allele.

During the course of the establishment of the TB-3a stocks discussed above, new information on the nature of the interaction of et and M^{et} has become available.

Crosses designed to introduce the modifier (M^{et}) into the TB-3a background are expected to produce kernels which develop into either of three types of plants with respect to their chromosome three constitution: (1) normal 3/3, (2) hypoploid $3/3^{B}$, and (3) hyperploid $3/3^{B}/B^{3}/B^{3}$.

Table 1
Expected genotypes (chromosomal and genic)
from the cross:

3 (a Et) /3 (a et) ; M^{et} M^{et} ♀ X 3 (A Et) /3^{B}/B^{3} (A Et) ; $M^{et}M^{et}$ ♂

All individuals are $M^{et}M^{et}$

Normal disjunction:

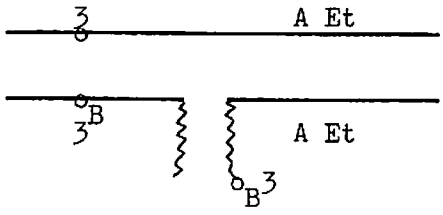
Seedling	Embryo Genotype	Endosperm Genotype	Kernel Phenoty
A Norm. green	3(a et) /3(A Et)	3(a et) /3(a et)/3(A Et)	A
A Norm. green	3(a Et)/3(A Et)	3(a Et)/3(a Et)/3(A Et)	A
A TB-3a green balanced	3(a et)/3^{B}/B^{3}(A Et)	3(a et)/3(a et)/3^{B}/B^{3}(A Et)	A
A TB-3a green balanced	3(a Et)/3^{B}/B^{3}(A Et)	3(a Et)/3(a Et)/3^{B}/B^{3}(A Et)	A

Nondisjunction:

A Hyper. green	3(a et)/3^{B}/B^{3}(A Et)/ B^{3}(A Et)	3(a et)/3(a et)/3^{B}	a et (Type I)*
A Hyper. green	3(a Et)/3^{B}/B^{3}(A Et)/ B^{3}(A Et)	3(a Et)/3(a Et)/3^{B}	a Et (Type II)*
a Hypo. vires.	3(a et)/3^{B}	3(a et)/3(a et)/3^{B}/ B^{3}(A Et)/B^{3}(A Et)	A (Type III)*
a Hypo. green	3(a Et)/3^{B}	3(a Et)/3(a Et)/3^{B}/ B^{3}(A Et)/B^{3}(A Et)	A (Type IV)*

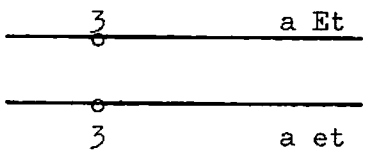
*Localization of lethal effect will be accomplished by comparing frequencies of Types I, II, III, and IV.

Balanced TB-3a
(pollen 15-20% aborted)



+/+ ♂ X

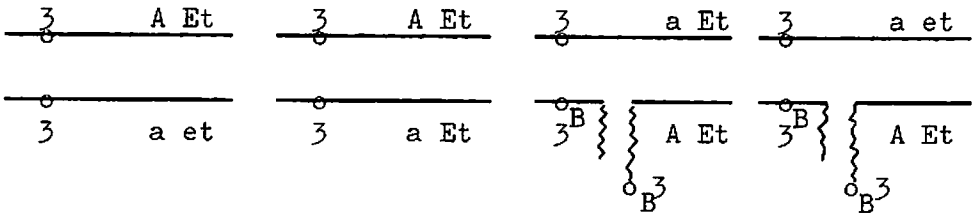
Normal 3/3
(pollen normal)



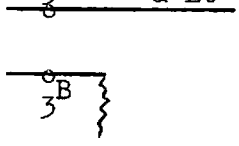
M et M et ♀ +

PROGENY: All individuals are +/M et

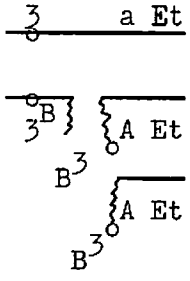
Normal ploidy
Nonvirescent:



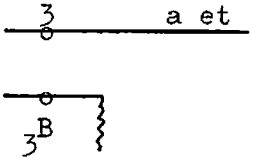
*Hypoploid
Nonvirescent:



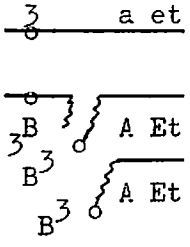
Hyperploid
Nonvirescent:



*Hypoploid
Virescent:



Hyperploid
Nonvirescent:



The mutant etched allele is pleiotropic and expresses itself as an "etching" of the endosperm of homozygous kernels as well as producing a virescence in the seedling stage. As is well known, chromosome three hypoploids are easily recognized by their short stature, pointed leaves, and 50% pollen sterility. Such a cross as the one just described is expected to produce equal numbers of nonvirescent hypoploids (3(a Et)/3^{B}) and virescent hypoploids (3(a et)/3^{B}). The results of field scoring of seedlings for two summers appear in Table 2.

Table 2

Results of seedling tests of progeny of the cross: $3(\underline{a}\ \underline{Et})/3(\underline{a}\ \underline{et});\underline{M}^{et}\underline{M}^{et}$ ♀ X $3(\underline{A}\ \underline{Et})/3^{B}B^{3}(\underline{A}\ \underline{Et})$; $+/+$ ♂. All individuals are $\underline{M}^{et}/+$.

	Normal Green Aa	Hypoploid Green aa	Hypoploid Virescent aa
1966 (U. of Ill.):			
No. of seedlings	1025	220	107
Frequency (%)	75.8	16.2	8.0
1967 (Ill. St. Univ.):			
No. of seedlings	479	125	56
Frequency (%)	72.5	19.0	8.5

It is obvious from the data that the two types of hypoploids are not present in equal frequencies as was the expectation. The degree of virescence associated with the deficient class (aa virescent) was extreme, bordering on albinism even though they eventually gained pigment. Further, these plants were only about 20% the size of the aa green hypoploids and formed on unexpected third category of plants with respect to size and rate of maturation. At maturity (though they never were observed to shed pollen), they were quite small resembling more fox tail grass than corn (though corn they were).

It is important to remember that all of these individuals are heterozygous for the modifier, $\underline{M}^{et}/+$. We consider this observation to be a reflection of an "enhancement" of the etched phenotype which is caused by the interaction of the mutant etched allele and the modifier $\underline{M}^{et}$.

Further evidence of such enhancement effects is available but not previously noted. As a result of conversations with Dr. I. Greenblatt (see also M.G.C.N.L. 36 & 37) I have become interested in the development of the etched kernel phenotype. While screening kernels for possible use in developmental studies, I made the purely subjective observation that etched kernels of the $+/\underline{M}^{et}$ background consistently have a more severe "etching" of their endosperm tissues than do etched kernels lacking the modifier. This past year etched kernels that were heterozygous for the modifier were planted in the field. Etched (et/et) kernels lacking the modifier were also planted. The seedlings were strikingly different with respect to their expression of virescence. Those plants that were heterozygous for the modifier displayed a level of virescence that borders on albinism. Those plants lacking the modifier had a "normal" level of

virescence. The severely virescent plants did, however, develop pigment and produced kernels which were used in a histological study.

These observations indicate that there is indeed an enhancement effect associated with the interaction of et and M^{et}. It apparently affects the development and maturation of plastids (chloroplasts and leucoplasts) as first suggested by Greenblatt (M.G.C.N.L. 36 & 37).

If we try to reconstruct this system in terms of enhancement effects and the postzygotic lethality associated with the etched locus (see Cox M.G.C.N.L. 40:39-42), the following picture emerges.

	Kernel Genotype	Endosperm Phenotype	Seedling Phenotype
I.	et/et; + +	Moderate to poor etching	Virescence +
II.	et/et; M^{et} +	Severe etching	Extreme virescence
III.	et/et; M^{et} /M^{et}	Postzygotic arrest, no mature kernels	---------

The above scheme suggests that the modifier, M^{et}, which is independent of the linkage group of the mutant etched allele, interacts with etched to upset normal plastid development.

The author would like to thank the laboratory of Dr. J. R. Laughnan and the Department of Agronomy, University of Illinois for the use of their field facilities during the summer of 1966. Our current work is being carried out at the Illinois State University Farm, Normal, Illinois where space has been set aside, largely through the efforts of Dr. D. F. Weber, for the establishment of maize genetics research plots.

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3. The effect of abnormal chromosome 10 on recombination in Tp9/N9 plants.

Rhoades (1958, M.G.C.N.L. 32:66-70 and unpublished) has intensively studied a segment transposed from the long arm of chromosome 3 (3L) to the short arm of chromosome 9 (9S). Recombination values were determined in bivalents consisting of one normal chromosome 9 (N9) and one carrying the transposed segment from chromosome 3 (previously designated as Dp9 but now designated as Tp9 by Rhoades). Recombination along the entire length of 9S was strongly decreased in plants heterozygous for the transposition. A corresponding decrease in the precision of chromosome pairing in 9S at pachynema has also been demonstrated by Rhoades and Dempsey (unpublished). They found that the buckle induced by the transposition is not located at a constant position in 9S, but it could be found at essentially any position in 9S. Frequently the buckle is not even seen since it was retracted into the chromosome. These genetic and cytological results have been confirmed by the author. (The same transposition was used by the author in a previous study reported in this newsletter (Weber, M.G.C.N.L. 41:204-206).

The present study is designed to determine the effect of abnormal chromosome 10 on this system. Recombination in sister plants of the following constitutions was analyzed: