Table 1

Aleurone color segregation in crosses of $(\frac{r\text{-}co}{r})$, F-co* x r r. (The r r tester was wxwx).

Cross	Non-variegated	Variegated	Total	X^2
3 0334-1 x 808	350	107	457	0.61 ns
3 0334-2 x 515	278	105	383	1.19 ns
3 0334-3 x 516	282	78	360	2.13 ns
3 0334-4 x 511	204	80	284	1.52 ns
3 0334-5 x 921	277	93	370	0.00 ns

Test for relation to the En system: In another series of crosses, variegated kernels of the Colombia line were crossed to $a_1^{m(r)}/a_1^{m-1}sh_2$, an En tester stock (Peterson, 1965, Amer. Nat. 99:391). The resulting F_1 was testcrossed by $a_1^{dt}\ sh_2/a_1^{dt}\ sh_2$ (a_1^{dt}, an allele that responds to Dt producing colored dots on a colorless background; sh_2 is a recessive allele conditioning shrunken endosperm and is very closely linked to the A_1 locus). Kernels with colored spots or sectors were obtained and the resulting plants were backcrossed to $a_1^{m(r)}/a_1^{m-1}sh_2$; the kernels on each of five ears obtained from the above cross were counted and grouped according to their phenotypic appearance (Table 2).

None of the X^2 values was significant at the .05 level of probability. Since the heterogeneity X^2 (14.61) was not significant, the data were pooled over all crosses and a non-significant X^2 value of 1.34 was obtained.

Tests to determine whether F-co is a Dt allele are presently in progress.

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3. T-cytoplasm mitochondrial membrane activities.

In view of the striking effect reported by Miller and Koeppe (1971) of Helminthosporium maydis race T toxin in causing the immediate uncoupling of oxidative phosphorylation and irreversible swelling in KCl

medium of T (Texas) but not of N (Normal) mitochondria, a study was initiated to investigate the various details of the pathways of electron transport and associated activities in N and T mitochondria. On the basis of the details of the pathways in Jerusalem artichoke mitochondria developed by Coleman and Palmer (1972, Eur. J. of Biochemistry 26:499), the effects of the race-T pathotoxin on various steps in this network of enzyme reactions was investigated. The race T pathotoxin causes an increase in the activity of cytochrome oxidase and succinate cytochromic reductase, possibly due to a disturbance of the mitochondrial membranes which allows increased substrate accessibility acting as an uncoupler.

The first ATP-coupled site of the electron transport chain, which includes the endogenous NADH dehydrogenase, was studied using malate as a substrate in the absence of exogenous NAD^+. In T mitochondria, the pathotoxin strongly inhibited the oxidation of malate by intact mitochondria. Malate oxidation via endogenous NADH dehydrogenase in N mitochondria was unaffected by similar concentrations of pathotoxin. Upon the addition of NAD^+, however, there is a marked stimulation of malate oxidation in intact T mitochondria. Thus, the presence of an intermembrane malate dehydrogenase activity coupled to NAD^+ reduction leads to an initial and immediate stimulation of malate oxidation via the exogenous NADH dehydrogenase. This confirms that the inhibition of malate and oxoglutarate oxidation in T mitochondria by pathotoxin is almost certainly at the endogenous NADH dehydrogenase complex of the inner membrane.

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4. Location of pg^m of the En system.

pg^m (Peterson, 1960 Genetics 45:115) has been found to be allelic with a pg^m isolated by Neuffer in mutagen treatments. This is uncovered by TB-3b, which places pg^m on chromosome 3S.

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