Table 1

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

<table>
<thead>
<tr>
<th>Cross</th>
<th>Non-variegated</th>
<th>Variegated</th>
<th>Total</th>
<th>(X^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 0334-1 x 808</td>
<td>350</td>
<td>107</td>
<td>457</td>
<td>0.61 ns</td>
</tr>
<tr>
<td>3 0334-2 x 515</td>
<td>278</td>
<td>105</td>
<td>383</td>
<td>1.19 ns</td>
</tr>
<tr>
<td>3 0334-3 x 516</td>
<td>282</td>
<td>78</td>
<td>360</td>
<td>2.13 ns</td>
</tr>
<tr>
<td>3 0334-4 x 511</td>
<td>204</td>
<td>80</td>
<td>284</td>
<td>1.52 ns</td>
</tr>
<tr>
<td>3 0334-5 x 921</td>
<td>277</td>
<td>93</td>
<td>370</td>
<td>0.00 ns</td>
</tr>
</tbody>
</table>

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 \(a_1^d\ sh_2/a_1^d\ sh_2\ (a_1^d\), an allele that responds to \(D\) 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 \(D\) allele are presently in progress.

Jaime Gonella
Peter A. Peterson

3. T-cytoplasm mitochondrial membrane activities.

In view of the striking effect reported by Miller and Koepp (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.

Peter A. Peterson
Richard B. Flavell
D. H. P. Barratt

*Plant Breeding Institute, Cambridge, England

4. Location of pgᵐ of the En system.

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

Peter A. Peterson