the restoration of fertility for any of the male-sterile cytoplasms and, consequently, do not appear to be the products of nuclear restorer genes. Finally, in a few instances, particular bands were observed to vary in only one cytoplasmic version of one inbred line. These may be the result of specific nuclear-cytoplasmic interactions. Protein bands that consistently varied between the cytoplasmic versions of these inbreds were not observed.

P. L. Bolen and E. H. Coe, Jr.

Electrophoresis of shoot and tassel mitochondrial proteins from diverse cytoplasms

Important considerations for the identification of specific gene action are the tissue and stage of development at which this action would be expected to occur. Rf3, the standard restorer for cms-S cytoplasm, is known to be expressed after the formation of the microspore (Buchert, J. G., Proc. Nat. Acad. Sci. USA 47:1436-1440, 1961). This fact suggests that Rf3 may produce a protein product just after microspore formation that is not present in microspores with rf3.

If mitochondria are the determinants of cytoplasmic male sterility and the Rf3 allele acts to correct this defect, then a consistent difference in mitochondrial isolates from post-meiotic tassels should be detectable between cms-S versions of two inbreds that possess different alleles (Rf3 and rf3) at this locus.

To investigate this hypothesis, mitochondria were isolated from shoot and tassel tissues of maintainer, cms-S, Wn and 249 cytoplasmic versions of K55 (non-restorer of cms-S) and Tr (restorer of cms-S) inbreds. The same two electrophoretic techniques were used to separate the mitochondrial proteins as in the preceding report.

A comparison of the banding patterns from shoot and tassel mitochondria isolated from the same line revealed developmental differences in protein constituents. Protein bands that were present in only shoot mitochondria and, reciprocally, bands that were present in only tassel mitochondria were identified. However, it was apparent that none of these developmental differences were the result of post-meiotic action by the Rf3 gene since a band present in only the tassel patterns of the various cytoplasmic versions of the inbred Tr (restorer of cms-S) was not identified.

P. L. Bolen and E. H. Coe, Jr.

Renaming of B-A translocations

I propose three changes in designation of translocations between B chromosomes and members of the basic set:

1. Call them B-A translocations instead of A-B translocations, since the B is normally listed first (e.g., TB-1a). Listing the A chromosome first (T1-Ba) would tend to obscure the uniqueness of these translocations.

2. Incorporate L or R into the name of each B-A translocation to indicate the A chromosome arm involved. TB-1a would thus become TB-1La; TB-1b would become TB-1Sb. This change should increase convenience in use.

3. Rename the compound translocations of F. A. Rakha and D. S. Robertson (Genetics 65:223) and of Robertson (NL 49:79) to indicate the B-A translocation originally used to develop each. TB-4L, 1L(4692) would thus become TB-1La-4L4692. Since a portion of the long arm of chromosome 1 is uncovered by this compound translocation, it is important to include 1L in the name. And since a portion of 4L is attached to the B1L chromosome, the order should be 1L-4L instead of the reverse. Concurrence of E. H. Coe, Jr., J. L. Kermicle, R. J. Lambert, Bor-yaw Lin, and D. S. Robertson with the changes outlined above has been obtained.

J. B. Beckett