some bt2 mutants (i.e., bt2-7315 and bt2-B) when Bt2 cDNA was used as probe (Figure 2). If Bt2-like were expressed routinely in the endosperm, then there would be Bt2-like transcript in all bt2 mutants. It thus appears that Bt2-like is not expressed in the wild type endosperm.

Since the transcript in bt2-7503 hybridizes strongly to a Bt2 probe and is larger than that encoded by the wild type Bt2 gene (Figure 2), the possibility that the bt2-7503 transcript comes from Bt2-like gene would require a double mutation; a mutation to abolish Bt2 transcript and a second mutation to turn on the Bt2-like gene in the endosperm. While not impossible, it is improbable. The fact that there are two sequence polulations in the duplicated region of bt2-7503 genomic DNA and mRNA suggests the possibility of both genes (bt2-7503 and bt2-like) being expressed in bt2-7503 endosperm.

It thus appears that the possible templates used in the amplification was the mRNA of bt2-7503 or mRNA of bt2-7503 and bt2-like.

Assuming the duplication occurred in bt2-7503, either the duplication in the 314bp intron or the import of intron sequence through non-homologous recombination between Bt2 and Bt2-like or both events caused the mutational lesion in bt2-7503. It is not clear which event occurred first, duplication or recombination.