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BREAKTHROUGHS AND VIEWS

Alternative Forms of β-pol mRNA Are Not Tumor-Specific and Are Not the Result of Mutations in the DNA

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DNA polymerase β plays an essential role in the DNA repair process. It is of great significance to determine whether some specific isoforms of this enzyme are associated with carcinogenesis. Bhattacharyya *et al.* (1) studied the expression of a DNA polymerase β isoform, carrying an 87-bp deletion. They claim that the expression of this isoform of DNA polymerase β, which they observed on the mRNA and protein level is due to a heterozygous alteration of the DNA polymerase β gene and is tumor-specific. However, such a change in the DNA polymerase β gene has never been shown before, and we cannot find any proof for it in this work. A previously published paper (2) strongly suggests that the 87-bp deletion arises from alternative splicing of DNA polymerase β mRNA rather than a deletion in the gene.

Bhattacharyya *et al.* (1) found this variant form of β-pol cDNA only in tumor samples which is in conflict with previously published work (3, 4) that showed its presence in both normal and tumor tissues. They are also wrong in claiming that different isoforms of β-polymerase mRNA were seen only in tumor cells or in the blood of Werner syndrome patients, as they have also been reported in normal cells:

1. The exon II deletion variant was seen in normal cell lines (2);
2. The exon α insertion (between exons VI and VII) variant has been detected in all cells tested (4);
3. The intron inclusion (between exons IX and X) variant has been observed in the blood of healthy donors as well as in Werner patients (5).

It may be concluded that alternative splicing of DNA polymerase β mRNA is not tumor specific and is ubiquitous.

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