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2 heads are better than 1: 2 dysfunctional DNA repair pathways kill tumor cells

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Individuals who inherit two mutant copies of any one of about 12 genes that make the proteins of the Fanconi Anemia (FA) pathway develop FA, which is characterized by increased incidence of cancer and bone marrow failure, among other things. However, individuals with just a single mutant copy of one of these genes are also at increased risk of developing cancer.

This occurs when the remaining "good" copy of the gene becomes mutated in a specific cell type, allowing that cell type to form a tumor. However, hope of a new treatment for these cancers has now been provided by researchers from the Dana-Farber Cancer Institute in Boston who suggest that inhibiting the protein ATM might kill these cancer cells.

In the study, which appears online on April 12 in advance of publication in the May print issue of the *Journal of Clinical Investigation*, Alan D'Andrea and colleagues show that loss of ATM function in human cell lines with a dysfunctional FA pathway caused the cells to die.

The dying cells were characterized by high levels of DNA breakage, which is consistent with the fact that FA pathway proteins and ATM are important regulators of two distinct DNA repair pathways. It therefore seems that the ATM pathway of DNA repair keeps the FA pathway–deficient tumor cells alive and that loss of this pathway results in tumor cell death. As FA pathway–deficient tumor cells were shown to be sensitive to an inhibitor of ATM, the authors suggest that ATM might

provide a therapeutic target for the treatment of individuals with FA pathway–deficient tumors.

Source: Journal of Clinical Investigation

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