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MAIN FIELDS OF RESEARCH; ABSTRACT

Fanconi anemia complementation group J (FANCJ) is one out of 21 proteins associated with Fanconi Anemia (FA). The cancer-prone syndrome FA was shown to cause hypersensitivity to agents inducing DNA inter-strand cross-links (ICL). Moreover, mutations in *FANCJ* are also associated with hereditary breast and ovarian cancers, which often derive from defects in the same DNA repair genes, such as *FANCD1 (BRCA2), FANCN (PalB2)*.

Given that FANCJ is involved in ICL and double-strand break (DSB) repair, it is considered as a tumor suppressor protein. FANCJ is an ATP-driven DNA helicase with a 5'-3' polarity and coordinates an iron-sulfur (FeS) cluster. FeS clusters are inorganic cofactors found in a wide variety of proteins carrying out fundamental biological processes. It was suggested previously that the FeS cluster of FANCJ is essential for its activities, however the exact function of the FeS cluster in FANCJ is so far unknown. Several patient mutations causing early-onset breast cancer or Fanconi anemia were found within the region coordinating the FeS cluster, indicating the importance of the FeS cluster for proper FANCJ function.

We use a combination of cellular biology and biochemistry to study the role of the FeS cluster in FANCJ and the impact of patient mutations on FeS cluster binding and FANCJ activity. In the long run, this study might help to design targeted therapies for patients harboring a mutation within the FeS coordinating region of FANCJ.

SPECIAL TECHNIQUES AND EQUIPMENT
Protein purification, biochemical characterization of enzymes, cell culture techniques, DNA fiber assa