RAD51 as a potential surrogate marker for DNA repair capacity in solid malignancies

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Targeting deficient mechanisms of cellular DNA repair still represents the basis for the treatment of the majority of solid tumors, and increased DNA repair capacity is a hallmark mechanism of resistance not only to DNA-damaging treatments such as cytotoxic drugs and radiotherapy, but also to small molecule targeted drugs such as inhibitors of poly-ADP ribose polymerase (PARP). Hence, there is substantial medical need for potent and convenient biomarkers of individual response to DNA-targeted treatment in personalized cancer care. RAD51 is a highly conserved protein that catalyzes DNA repair via homologous recombination, a major DNA repair pathway which directly modulates cellular sensitivity to DNA-damaging treatments. The clinical and biological significance of RAD51 protein expression is still under investigation. Pre-clinical studies consistently show the important role of nuclear RAD51 immunoreactivity in chemo- and radioresistance. Validating data from clinical trials however is limited at present, and some clinical studies show controversial results. This review gives a comprehensive overview on the current knowledge about the prognostic and predictive value of RAD51 protein expression and genetic variability in patients with solid malignancies.

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