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Sommario/riassunto	Poly-ADP ribose polymerase (PARP) proteins are critical mediators of DNA repair. Many traditional anti-cancer chemotherapy agents overwhelm a cell’s ability to repair DNA damage in order to kill proliferating malignant cells. Recent evidence suggests that cancers within and across tissue types have specific defects in DNA repair pathways, and that these defects may predispose for sensitivity and resistance to various classes of cytotoxic agents. Breast, ovarian and other cancers develop in the setting of inherited DNA repair deficiency, and these cancers may be more sensitive to cytotoxic agents that induce DNA strand breaks, as well as to inhibitors of PARP activity. A series of recent clinical trials has tested whether PARP inhibitors can achieve synthetic lethality in hereditary DNA repair-deficient tumors. At the current time, mutation of BRCA serves as a potential, but not comprehensive, biomarker to predict response to PARP inhibitor therapy. Mechanisms of resistance to PARP inhibitors are only recently being uncovered. Future studies seek to identify sporadic cancers that harbor genomic instability rendering susceptibility to PARP inhibitors that compound lethal DNA damage.