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THE ROLE OF DICER IN DNA REPAIR AND COLON CANCER CHEMOTHERAPY

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Dicer is the key component of the RNA interference pathway. In 2008, our group reported that Dicer knockdown led to DNA damage accumulation in mammalian cells. Subsequently, two groups showed that Dicer-dependent small RNAs produced from the sequences in the vicinity of DNA double-strand break (DSB) sites were essential for homologous recombination-mediated DSB repair. Recently, we found that Dicer is associated with SIRT7 and is required for DNA damage-induced chromatin relaxation by promoting H3K18Ac deacetylation, decreased Dicer expression inhibited non-homologous end joining by preventing chromatin relaxation at DSB sites. Moreover, we demonstrated that Dicer knockdown and overexpression increased and decreased respectively, the chemosensitivity of colon cancer cells, and that Dicer protein expression in colon cancer tissues of patients was directly correlated with chemoresistance. Our findings suggest that manipulation of Dicer expression may improve chemotherapy effects for patients with colon cancer, and possibly other cancers.

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