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Brca2 C-terminus interacts with Rad51 and contributes to nuclear focus formation in double-strand break repair of DNA

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ABSTRACT

In humans and mice, the interaction between the breast cancer susceptibility protein, BRCA2, and RAD51 recombinase is essential for DNA repair by homologous recombination, the failure of this process can predispose to cancer. Cells with mutated BRCA2 are hypersensitive to ionizing radiation (IR) and exhibit defective DNA repair. Using yeast and mammalian two-hybrid assays, we demonstrate that canine Rad51 protein interacts specifically with the C-terminus of canine Brca2. In support of the biological significance of this interaction, we found that radiation-induced focus formation of Rad51 in COS-7 cells was compromised by forced expression of the C-terminus of canine Brca2. A similar result was obtained for the murine C-terminus. These data suggest that the C-terminal domain of canine Brca2 functions to bind Rad51 and that this domain contributes to the IR-induced assembly of the Rad51 complex *in vivo*.

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