Identified in a genetic screen for mutants that are sensitive to ionizing radiation, RAD55 is a member of the RAD52 epistasis group. Other members of this group include RAD50, RAD51, RAD52, RAD54, RAD57, RAD59, MRE11, and XRS2. All members of the RAD52 epistasis group are involved in the repair of double-stranded breaksin DNA. Mutants are defective in the repair of DNA damage caused by ionizing radiation and MMS, in the maintenance of telomere length, in mitotic and meiotic recombination, and in mating-type switching because DSB intermediates are involved in these processes.Rad55p functions as a heterodimer with Rad57pto promote the formation of the Rad51p:single-stranded DNAnucleoprotein filament at the site of a DSB by displacing Replication Protein Afrom ssDNA. Formation of the Rad51p:ssDNA filament is required for homology searching and strand exchange during DSB repair via homologous recombination. A Rad51p mutant with a higher ssDNA binding affinity bypasses the need for the Rad55p-Rad57p heterodimer. Deletion of RAD51 or overexpression of Rad51p suppresses the cold sensitivity phenotype of a rad55 rad57 double mutant. Rad52p also helps assemble Rad51p onto ssDNAbut both Rad52p and the Rad55p-Rad57p heterodimer are required for efficient assembly of Rad51p at sites of DSBs during mating-type switching, meiosis, and mitosis.The Rad55p-Rad57p heterodimer is regulated by DNA damage checkpoints. Rad55p is phosphorylated by the checkpoint kinase Rad53p in response to DNA damaging agents such as methyl-methane sulfonate, gamma ray radiation, and UV radiation, as well as collapsed replication forks.Rad55p and Rad57p are paralogs of S. cerevisiae Rad51p. RAD51 paralogs in other organisms include Rhp55 and Rhp57 in S. pombe, AtXRCC3 and AtRAD51C in A. thaliana, and XRCC2, XRCC3, Rad51B, Rad51C, and Rad51D in humans and other vertebrates.