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Genetic instability is one of the hallmarks of cancer cells. Mutations in genes that are required for repairing DNA double strand breaks (DSB) have been implicated as a potential source of genetic instability and can promote the development of cancer. The importance of these processes is underscored by the fact that the abnormal repair of DSBs leads to the type of genetic alterations frequently observed in cancer cells. Furthermore, several inherited human diseases, which are characterized by immune dysfunction and predisposition to cancer, result from mutations in DSB repair genes. All organisms have therefore evolved an intricate network of systems for repairing their chromosomal breaks. The two highly conserved mechanisms for repairing DSBs are homologous recombination (HR), where the break is repaired by recombination between homologous sequences, and non-homologous end-joining (NHEJ), where little or no homology is required to join the ends of the DNA. The goal of our research is to elucidate the molecular mechanisms of non-homologous end joining (NHEJ), one of the two most important DSB repair pathways in eukaryotes. Additionally, we are beginning to unravel genetic requirement for a novel mechanism for repairing DSBs using a *Saccharomyces*-based genetic assay and for channeling DSBs to specific repair paths.