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Making way for repair

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In the 21/28 December Nature Downs *et al*. suggest that phosphorylation of budding yeast histone H2A in response to radiation damage loosens up chromatin to allow the entry of repair proteins (*Nature* 2000, **408**:1001-1004). They mutate the carboxyl terminus of H2A to replace a serine, whose phosphorylation is dependent on the DNA-damage-induced kinase Mec1, with an alanine. The resulting strain is sensitive to agents that induce double-strand breaks (DSBs), but not to ultraviolet light or mutagens that affect a single DNA strand. Transcriptional responses to DNA damage, checkpoint delays, and repair by homologous recombination are not affected by the mutation, but DSB repair by non-homologous end joining (NHEJ) is defective. A serine to glutamic acid substitution that mimics the phosphorylated H2A results in plasmids that are topologically more relaxed, and regions of DNA between individual nucleosomes that are more susceptible to degradation. Thus the Mec1-dependent phosphorylation of H2A may facilitate NHEJ by decondensing chromatin to allow repair proteins better access to the DNA.

References

1. Nature, [http://www.nature.com/nature/]

2. Crystal structure of the nucleosome core particle at 2.8 A resolution.