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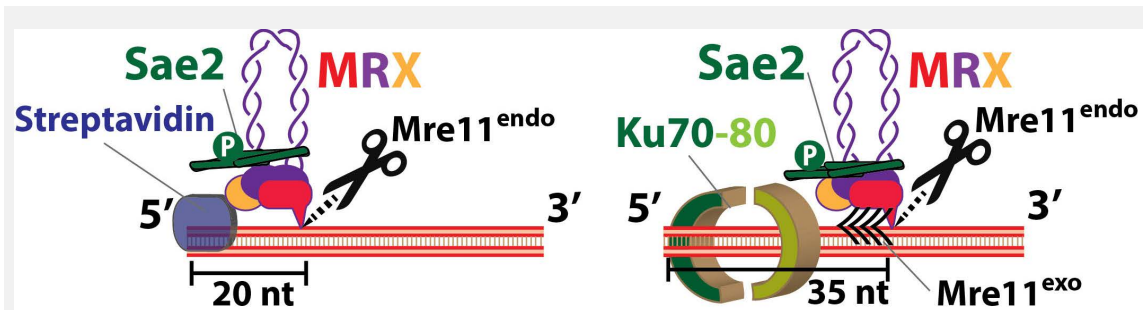
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A new paper from the Cejka lab published in Genes & Development

on Tuesday, January 9, 2018

A paper published today in the journal of Genes & Development describes how cells repair broken DNA. DNA of all living organisms is prone to breakage, and a failure to faithfully repair broken DNA may lead to cell death, mutagenesis and cancer in high eukaryotes. Cells use either the homologous recombination or non-homologous end-joining pathway to repair DNA breaks. Giordano Reginato, Elda Cannavo and Petr Cejka from the IRB, an institute affiliated to the Università della Svizzera italiana, use the budding yeast *Saccharomyces cerevisiae* as a research model to investigate the mechanisms of DNA break repair.

In 2014, the Cejka laboratory described that the Mre11-Rad50-Xrs2 protein complex, together with phosphorylated Sae2, cleave one DNA strand in the vicinity of DNA breaks to initiate DNA repair by the homologous recombination pathway (Cannavo et al., Nature, 514, 122-5). In the current study, the IRB team shows that proteins that are highly abundant at DNA ends, such as the non-homologous end-joining protein Ku, promote DNA cleavage by the Mre11-Rad50-Xrs2 and Sae2 machinery. This describes the interplay between the two key DNA double strand break repair pathways. The results show that Ku can also promote DNA end processing and therefore recombination. The research communication is published together with a separate [paper](#) from the Sung laboratory (Yale University) that describes similar results. L. Symington (Columbia University) wrote a [commentary](#) that highlights both papers.



To initiate repair of broken DNA by homologous recombination, the repair machinery must first endonucleolytically cleave the 5'-terminated DNA strand at the break site. The Mre11-Rad50-Xrs2 (MRX) complex together with Sae2 has such activity, but must be stimulated by protein blocks such as non-physiological streptavidin. Reginato, Cannavo and Cejka now show that the Ku heterodimer, a highly abundant protein that binds DNA ends with a high affinity, has the capacity to direct DNA cleavage by MRX.

Article

Physiological protein blocks direct the Mre11–Rad50–Xrs2 and Sae2 nuclease complex to initiate DNA end resection

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