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Webinar « Genome dynamics and epigenetics »

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RAD54 is essential for RAD51-mediated repair of meiotic DSB in Arabidopsis

Homologous recombination is a universal pathway which repairs broken DNA molecules through the use of homologous DNA templates. It is both essential for maintenance of genome stability and for the generation of genetic diversity through sexual reproduction. A central step of the homologous recombination process is the search for and invasion of a homologous, intact DNA sequence that will be used as template. This key step is catalysed by the RAD51 recombinase in somatic cells and the two DNA strand-exchange proteins RAD51 and DMC1 in meiotic cells, assisted by a number of associated factors. Among these, the chromatin-remodelling protein RAD54 is a required cofactor for RAD51 in mitotic cells. Understanding of its role during meiotic recombination has however remained elusive. Using a combination of genetic and cytogenetic approaches, we show that absence of RAD54 has no detectable effect on meiotic recombination in otherwise wild-type plants but becomes essential for meiotic double strand break repair by RAD51 in absence of DMC1. We further show that this function is downstream of the meiotic role of RAD51 in supporting the activity of DMC1. Our findings have several interesting implications for the regulation of the strand invasion step during meiotic recombination in plants, and very probably also other multicellular eukaryotes.


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