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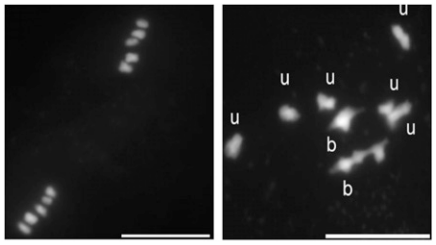
IN BRIEF

Embracing Diversity: Uncovering the Mechanism Underlying Interhomologous Recombination Bias during Meiosis

Do you want your children to be exactly like you? Didn't think so. Such a situation would impair the evolution of any organism, including plants, which usually reproduce sexually to adapt to an ever-changing environment. During meiosis, alleles are shuffled between homologous chromosomes (derived from both parents) via recombination, leading to new combinations of genetic traits in the following generation. Meiotic recombination in plants begins with the programmed induction of DNA double-strand breaks. Sections of DNA around the 5′ ends of the break are cut away, yielding an overhanging 3′-single-stranded DNA, which associates with protein factors, including the recombinases RADIATION SENSITIVE51 (RAD51) and DISRUPTED MEIOTIC cDNA1 (DMC1), to form a nucleoprotein filament. This structure allows the single-stranded DNA to invade a similar or identical DNA molecule that has not been broken, which serves as the template for DNA repair (Edlinger and Schlögelhofer, 2011). Despite the presence of a nearby sister chromatid, the meiotic DNA repair machinery is often directed to the homologous chromosome as the repair template, ensuring that the genetic material is sufficiently shuffled, thereby promoting diversity. While it is known that the HOMOLOGOUS-PAIRING PROTEIN2/MEIOTIC NUCLEAR DIVISION PROTEIN1 (HOP2/MND1) complex is crucial to this process in *Arabidopsis thaliana*, since loss of either MND1 or HOP2 results in the failure of DNA repair, the molecular mechanisms underlying the promotion of interhomologous recombination (and the possibly correlated suppression of intersister recombination) are currently unclear.

Uanschou et al. (pages 4924–4940) identified some highly informative mutants that helped uncover the process underlying interhomologous recombination bias in *Arabidopsis*. These mutants, *hop2-2* and *hop2-3*, have short siliques and a reduced number of seeds but otherwise appear normal. On closer inspection, they exhibit strong abnormalities in meiotic progression. During male meiosis, normal chromosome pairing does not occur in prophase, and very few bivalents (but numerous univalents) are observed among *hop2-2* metaphase cells (see figure). Segregation at meiosis II yields unbalanced pools of chromosomes at the end of meiosis; thus, four haploid daughter cells are not always produced, leading to abnormal pollen and, ultimately, fewer seeds. It turns out that in these mutants, RAD51 and DMC1 are loaded onto the nucleoprotein filament as usual but only the intersister DNA repair pathway is activated, making *hop2-2* and *hop2-3* excellent tools for the study of interhomologous recombination bias. The *hop2-2* phenotype is correlated with a decrease in HOP2/MND1 complex abundance, while the *hop2-3* mutant contains truncated HOP2 protein, which binds to MND1 in an inefficient manner compared with wild-type HOP2. Therefore, both mutants have less available functional HOP2/MND1 complex than the wild type, which makes it more difficult for DMC1 to enable repair via the homologous chromosome. By contrast, genetic analysis demonstrated that HOP2/MND1 is dispensable for RAD51-mediated intersister DNA repair. In addition, mutant analysis revealed that in the presence of DMC1 and in the absence of HOP2, RAD51-mediated repair is suppressed, suggesting that DMC1 is a negative regulator of RAD51 during meiosis. This negative regulation appears to be transient and is important for the establishment of DMC1-mediated, interhomolog connections during meiotic DNA repair. Thus, while RAD51 and DMC1 both form a nucleoprotein filament with broken DNA to initiate repair during meiosis, these proteins appear to have very different agendas. If RAD51 won out in wild-type plants, intersister repair would dominate and diversity would be lost.



At metaphase I, five bivalents always align on the metaphase plate in the wild type, while in *hop2-2*, a mixture of intact univalents and bivalents are observed. u, univalent; b, bivalent. Bars = 10 μm. (*Reprinted from Uanschou et al. [2013], Figure 1A*.)

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