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**Crossover control by mismatch detection protein MSH2 in response to the chromosome heterozygosity pattern in *Arabidopsis thaliana***

During meiosis, homologous chromosomes pair and reciprocally exchange genetic material in a process called crossover or meiotic recombination. Crossover is important for generating new allele combinations and at least one crossover per bivalent is necessary to ensure proper chromosome segregation in meiosis. Moreover, crossover placement is not random and its numbers are limited – there are only about 2-3 crossovers per chromosome pair per meiosis, regardless of the physical size of the genome.

One of the factors influencing recombination pattern is interhomolog polymorphism. The presence of heterozygous region juxtaposed to homozygous region on the same chromosome, causes a redistribution of crossovers into the polymorphic region. I showed that this heterozygosity juxtaposition effect depends on the activity of MSH2 protein, key element of mismatch repair system. My results demonstrate MSH2 stimulating role in the formation of Class I crossovers. With genome-wide crossover mapping in *msh2* hybrids, I was able to show that recombination is redistributed from highly polymorphic pericentromeres into less polymorphic subtelomeric regions. Genetic interference was not changed in msh2 inbred and hybrid lines.

By using a panel of *Arabidopsis thaliana* fluorescence-tagged lines (FTLs), I showed that recombination landscape is mostly similar between inbred and hybrid lines, however the presence of heterozygous region on an otherwise homozygous chromosome, redirects crossover events into the former. The increase in heterozygous region is mostly observed close to the heterozygous-homozygous regions boarder, whilst decrease in homozygous part spans the entire region. The protocol for FTL use in crossover rate measurements is also included in this dissertation.

Class II crossovers are polymorphism-sensitive and cannot be efficiently formed in heterozygous regions. In hybrids exhibiting only Class II (*fancm zip4* double mutant), crossover scarcity is the reason for reduced fertility. By additionally inactivating MSH2, I was able to increase plant fertility. Moreover, my genome-wide crossover analysis in different mutant contexts, including *msh2 fancm* and *msh2 recq4*, combined with FTL crossover frequency measurements, revealed that MSH2 limits Class II crossovers. Hence, MSH2 has opposite roles in two crossover pathways. In pericentromeric regions, which are much more polymorphic than the rest of the chromosome, MSH2 inactivation was not able to increase Class II crossovers frequency in heterozygous regions. This shows MSH2-independent polymorphism impact on recombination.

Finally, the overexpression of pro-crossover HEI10 caused significant increase in recombination in all tested heterozygosity variants, with a trend of heterozygous regions attracting crossovers still present. I showed, that in *msh2 HEI10-OE* total recombination is increased because of HEI10 promoting Class I, however no juxtaposition effect is observed. What is more, no HEI10 stimulation is detected in *msh2 fancm zip4 HEI10-OE* variant, proving that HEI10 has no role in Class II.

To sum up, I showed pro-recombination role of MSH2 for Class I crossovers and an antagonistic, anti-recombination role for Class II crossovers. Therefore, this work demonstrates that MSH2 is a master regulator of both crossover pathways, which allows for dynamic regulation of meiotic recombination outcomes, depending on the level and distribution of sequence divergence between homologs.