

Zip1 C-terminal Phosphorylation Promotes Zip1-Sgs1 Interaction in Meiotic Cells

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Meiotic recombination is essential to create novel allele combinations and ensure proper segregation during gametogenesis. Descriptions of detailed mechanisms of recombination are of interest because meiosis is a universal process that causes crippling human karyotypic disorders when faulty. Recombination in yeast involves three pathways, one of which involves Zip1, a protein that promotes interhomolog interactions enabling crossovers and synapsis. In mitotic cells, negative charges at S801-802 on Zip1 have been shown to facilitate interaction between Zip1 and Sgs1, a protein that disassembles homologous strand invasion intermediates. This study's goal was to characterize the importance of S801-802 phosphorylation in promoting Zip1-Sgs1 interaction within meiotic cells. Mutant zip1 plasmids were transformed into SGS1 and sgs1-meiotic depletion strains and analyzed for sporulation and meiotic progression. When S801-802 were mutated to alanines in Zip1 mutants defective for biased crossover formation, sporulation and meiotic progression worsened considerably; constitutively introducing negative charges by mutating S801-802 to aspartates partially rescued this phenotype. When Sgs1 was absent during meiosis, all three mutants exhibited meiotic arrest phenotypes, indicating that Sgs1 is the protein interacting with Zip1 to rescue recombination. This study identifies Zip1-Sgs1 interaction in meiotic cells, demonstrates that S801-802 phosphorylation mediates this interaction, and provides a mechanistic model for this interaction.

Awards Won:

Second Award of \$2,000

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