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Abstract

Chromosome miss-segregation during meiosis is a major factor contributing to birth defects as well as many genetic anomalies through the formation of aneuploid gametes, i.e. gametes with a deficit or surplus of one or several chromosomes. The synaptonemal complex (SC) is a major protein structure assembled with the synapsis of homologous chromosomes and is conserved from unicellular yeast to humans. One of its major roles during prophase I in meiosis is providing a structural framework for the maintenance of synapsis to facilitate the completion of reciprocal crossover events. Failure to form an effective SC or to experience crossover events leads to a cellular arrest in prophase I of meiosis. Through a genetic screen, our lab has identified a Nobel gene that suppresses cellular arrest in cells with specific mutations that cause chromosome mis-segregation. In our series of experiments we seek to garner a greater understanding of this Nobel gene's function in the bypass of similar mutations leading to a prophase I cellular arrest. In the model organism budding yeast (*S. cerevisiae*), the ZIP1 protein polymerizes to form the transverse filaments that assist in maintaining the stability of the SC. Studies of the synaptonemal complex have exposed an understanding of a relation between recombination and mis-segregation. A mutation of the Zip1 gene is an example of these anomalies resulting in meiotic cellular arrest delineating similar results to those produced by a deletion mutant of the strand invasion protein Dmc1. Our lab now demonstrates an effective suppression of the cellular arrest resulting from Zip1C1 as well as that produced by a deletion mutant of Dmc1. Further understanding of this Nobel gene and its pathway are being advanced at the present in the distinction of its relation to different proteins involved in the processing double strand breaks (DSB).