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Timing of meiosis: Microtubules on the move

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Meiosis represents a typical feature of the eukaryotic life style, which is tightly associated with sexual reproduction. Its fundamental role is the formation of haploid gametes from diploid germ-line cells. This process involves several crucial events orchestrated in the 2 rounds of meiotic divisions and requires the pairing and recombination of homologous chromosomes. The failure of proper chromosomal segregation is the major cause of chromosomal aneuploidy, resulting in spontaneous miscarriages and congenital birth defects in human.[1] As meiosis emerged early in eukaryotic evolution, its mechanism and corresponding genes are conserved across the eukaryotes.[2] In fission yeast, *Schizosaccharomyces pombe*, haploid cells with opposite mating types mate, their nuclei undergo karyogamy, and the diploid nucleus readily enters into meiotic program, resulting in the formation of four haploid spores. Two recent studies[3,4] point to the role of microtubule-associated proteins in proper timing of karyogamy and the first meiotic division, in which the chromosome number is reduced. During meiosis I, the chromosomal termini (telomeres) cluster at the nuclear envelope, resulting in a polarized configuration of chromosomes called the "bouquet" that plays a key role in coordinating the microtubule-organizing center and the spindle during meiosis.[5] The bouquet formation is followed by oscillatory movement of the elongated nucleus (termed "horsetail").[6] The telomere clustering, horsetail movement, and recombination are essential for homologous chromosome pairing in meiosis I.

In their report, Polakova et al.[3] screened the collection of *S. pombe* knockout strains for mutants with abnormal chromosome segregation. They identified 2 genes, *mal3*$^{+}$ and *mto1*$^{+}$, coding for microtubule plus-end-tracking protein and microtubule-organizing protein, respectively. The nuclei of *mal3*Δ and *mto1*Δ cells frequently enter into meiosis I before completion of karyogamy. Such twin divisions of haploid nuclei produce asci containing up to 8 spores. Interestingly, a fraction of spores resulting from aberrant meiosis remain viable. In addition, *mal3*Δ and *mto1*Δ mutants have impaired horsetail movement and proper segregation of homologous centromeres. Yamashita et al.[4] came to a similar conclusion and demonstrated that the defect in *mal3*Δ cells can be potentiated by mutation *dhc1*Δ or *ssm4*Δ coding for dynein heavy chain and a subunit of the dynactin complex, respectively. These authors also showed that the asci with supernumerary spores can also be generated from the wild-type cells by treatment with thiabendazole, a drug destabilizing microtubules. The results from both laboratories indicate that microtubule structures play a crucial role in coordination of meiotic events, although the details of mechanism controlling the completion of karyogamy before meiotic division remain elusive. Nevertheless, these studies provide solid ground for further investigations of this relatively unexplored area.

References

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