excitement about all of these questions and open a new era in the study of voltage-gated channels. Stay tuned to this new family channel.

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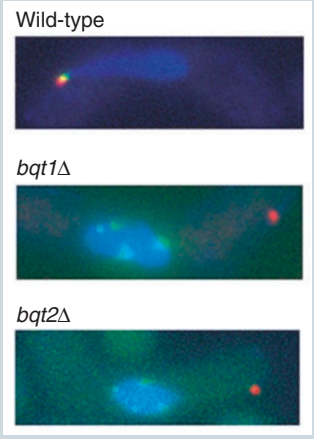
Gathering bouquets

Chromosome segregation involves pairing and recombination of homologous chromosomes. During the meiotic cell cycle, a single round of replication is followed by two consecutive nuclear divisions. Both events require major chromosomal movements, and missegregation during meiosis can result in developmental defects. During meiotic prophase, telomeres, which are usually spread along the nuclear envelope during the mitotic cycle, cluster together at the nuclear envelope. This telomere grouping results in a 'bouquet' arrangement of chromosomes that is characteristic in many organisms, suggesting a conserved role for telomeres in meiosis.



Schizosaccharomyces pombe is an amenable system for studying bouquet formation. During normal mitosis in *S. pombe*, the centromeres are associated with the spindle pole body (SPB). However, during meiosis, the chromosomes instead become tethered to the SPB through the telomeres. This telomere-SPB association has been shown to facilitate homologous chromosome pairing and recombination and is required for formation of the meiotic bouquet.

There are several proteins in both the telomeres and SPB known to be important in bouquet formation. Two of them, Rap1 (human RAP1) and Taz1 (human TRF1 and TRF2), are found constitutively associated with telomeres in *S. pombe*. Taz1 binds directly to the telomeric repeats and is required for Rap1 telomere binding. Although these proteins are non-essential for mitotic growth, *rap1* or *taz1* deletion strains have defects in meiotic telomere clustering. Sad1 is an SPB component crucial for mitotic growth, whereas Kms1 is an SPB component important in meiosis. Cells lacking Kms1 show defects in telomeric clustering: rather than forming a single spot at the SPB, Sad1 forms several distinct foci at the nuclear envelope, to which the telomeres localize.

Despite what is known about the importance of SPB and telomere components during meiosis, there is little information about what brings these complexes together in preparation for chromosome segregation. Now, Hiraoka and colleagues (*Cell* **125**, 59–69, 2006) have identified two novel proteins that are required to physically link the telomeres to the SPB during bouquet formation. Because the mating pheromone response induces meiotic telomere clustering in *S. pombe*, the authors looked for genes whose expression was induced in response to mating-pheromone signaling and systematically disrupted each of these genes. One strain showed loss of telomere clustering, and they named that gene *bqt1* (for bouquet formation). Disruption of a second gene, *bqt2*, identified by a two-hybrid screen using Bqt1 as bait, resulted in loss of bouquet formation during meiotic prophase. Although the authors found no obvious homologs for Bqt1 or Bqt2, Bqt1 shares weak sequence similarity with Dam1, a fungal centromere protein found associated with the kinetochore during mitosis.

Characterization of GFP-tagged Bqt1 and Bqt2 showed that both proteins colocalized with Taz1 and Sad1 at the telomere-SPB cluster during meiosis. Deletion of *bqt2* resulted in dispersed telomeres (green), with Bqt1 colocalized with Sad1 (red) at the SPB. In the absence of Bqt1, Bqt2 had diffuse nuclear localization and the telomeres were not localized at the SPB. Additional experiments defined interactions between telomere and SPB components, showing that Bqt1 binds directly to Sad1, whereas Bqt2 can only bind Sad1 when Bqt1 is present. Both Bqt1 and Bqt2 are required to interact with Rap1. Artificial expression of Bqt1 and Bqt2 in mitotic cells showed that both can recruit Sad1 to the telomeres, confirming that these two proteins are sufficient to bridge SPB and telomere components. Time-lapse imaging of meiotic cells shows that Sad1, initially localized at the SPB, transiently disperses to Rap1-bound telomeres and then returns to the SPB.

The authors propose a model for bouquet formation in which Bqt1 and Bqt2 recruit Sad1 to Rap1 at the telomeres along the nuclear envelope. Sad1 then brings the complex back to the SPB, where efficient chromosome pairing and recombination can occur. The authors suggest that Kms1, which has been shown to interact with the cytoskeletal motor protein dynein, may be responsible for bringing Sad1, and the attached telomeres, back to the SPB. This role would explain the Sad1 foci observed in Kms1-deficient cells. These studies provide new insight into how the cell performs the dramatic chromosomal movements associated with meiosis.

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