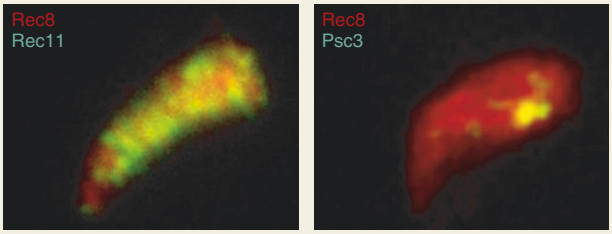
Recasting meiotic cohesion



Localization of Rec8 (red) and its partners Rec11/Psc3 (green) on chromosomes during meiotic prophase. Rec8 localizes along whole chromosome regions; however, Psc3 associates mainly at the clustered centromeres and Rec11 along the arm regions.

Chromosome segregation consists of a series of highly orchestrated events that are essential for cell division. A central aspect of this process is cleavage of cohesin, a multisubunit complex that maintains the structural integrity of chromosomes. Cohesin consists of two structural components, Smc1 and Smc3 (Psm1 and Psm3 in the fission yeast *Schizosaccharomyces pombe*, respectively), and two accessory components that are specific for mitotic or meiotic events. Meiosis poses a far more complex problem for chromosome cohesion than mitosis, as first reductional division of chromosome pairs at meiosis I must occur before a mitotic-like equational division at meiosis II. The meiotic cohesin complex is central to recombination events, and studies in fission yeast have identified Rec8 as a meiosis-specific accessory subunit that replaces Scc1, a subunit specific to the mitotic complex. During meiotic prophase, a Rec8-containing complex is localized along the entire chromosome, facilitating chromatid cohesion and recombination. Centromeric Rec8 ensures that each kinetochore of sister chromatid pairs is attached to the same spindle pole (monopolar attachment). Initially, Rec8 is only disrupted along chromatid arms during meiosis I. By meiosis II, however, Rec8 at centromeres is disrupted, allowing separation of sister chromatids. A good candidate for the second accessory meiosis-specific subunit is Rec11, which replaces the mitosis-specific Psc3 subunit. Mutation of Rec11, similarly to mutation of Rec8, reduces recombination. However, a direct demonstration of Rec11 function in the meiotic cohesin complex has been lacking.

Now, a study by Watanabe and colleagues (*Science* **300,** 1152–1155 (2003)) provides a more detailed molecular explanation of these events. Their first significant observation is that sister chromatids dissociate prematurely during meiosis I in *rec11Δ* cells. This suggests that Rec11 is central to sister chromatid cohesion. Using a similar approach, they also examine the role of Rec11 in centromere function and find that monopolar spindle attachment is dependent on Rec8, but not on Rec11. This raises the intriguing possibility that Rec11 is not the only binding partner of Rec8.

Enter Psc3. Analysis of *psc3* mutants demonstrate that it may have a minor role in cohesion along meiotic chromosome arms, but is central to kinetochore regulation at meiosis I and II. Here, the authors go further, demonstrating that two distinct Rec8–Psc3 complexes exist: the first complex is localized to pericentromeric regions and is dependent on the heterochromatic state of this region for its localization. In contrast, a second Rec8–Psc3 complex is localized to the central core and does not require heterochromatin for its localization. Interestingly, these differences in localization reflect distinct functions at different stages of meiosis: the pericentromeric complex is required for centromere cohesion until meiosis II, whereas the central-core complex is most probably required earlier, for monopolar attachment at meiosis I.

Thus, the authors envisage the following model of meiotic chromosome segragation: first, the Rec8–Rec11 complex is disrupted during meiosis I, allowing separation of sister chromatids along chromosome arms; second, the pericentromeric Rec8–Psc3 complex maintains chromosome cohesion until meiosis II, when its disruption results in complete dissocation of sister chromatids; third, the central-core Rec8–Psc3 complex establishes monopolar attachment of sister kinetochores.

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Neuronal polarization: building fences for molecular segregation

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Neurons exhibit distinct compositions in the axonal and dendrite plasma membrane, but it remains ambiguous whether or not a diffusion barrier is needed to keep the different components separated. Now, Nakada *et al.* utilize state of the art microscopy to follow the dynamics of single lipids or proteins inserted in different areas along the axonal and dendritic surface of neurons at different developmental stages. The results obtained shed new light on the mechanism underlying polarized segregation of membrane components in neurons.

The common neuron in the central nervous system contains three easily recognizable regions — the cell body, numerous tapered and highly branched dendrites and a single long axon that ramifies near the site of contact with the target organ. As pointed out more than a century ago by Ramon *y* Cajal, the neuron is polarized both structurally and functionally, with the dendrites and cell body receiving and processing information through the synaptic potential and the axon delivering information by generating and propagating an action potential. The functional differences between axons and dendrites are direct consequences of the distinct molecular compositions of both the cytoplasm and the plasma membrane[1].

Biologists working on neuronal polarity