

**P001** Premeiotic G1 to S transition and mutual inhibition of hotspots in *Schizosaccharomyces pombe* meiosis  
**Juerg Kohli, Eveline Doll, Benjamin Sakem**  
*Institute of Cell Biology, University of Berne, Switzerland*

Azygotic meiosis in  $h^+/h^-$  diploids is induced by nitrogen starvation. A systematic analysis of mutants of genes coding for proteins involved in meiotic sister chromatid cohesion, chromosome pairing, double-strand break (DSB) formation, and repair of DSBs, showed that the start of premeiotic S phase and of subsequent events is advanced for up to two hours in several mutants (eg *rec11*, *rec12*, *rec14*), like wild-type in a few mutants (*mde2*, *dmc1*), and delayed in one case (*rec8*). A preliminary model on premeiotic G1 to S transition will be presented. The meiotic recombination hotspot at *ura4A* was correlated with a double-strand break (DSB) 500 bp upstream of the gene. The dependence of *ura4A* hotspot activity and DSB formation on proteins modifying chromatin structure and enhancing the M26 hotspot was investigated. It was found that the *ura4A* hotspot is not dependent on Pcr1, which is required for hotspot activity at *ade6-M26*. Recombination analysis indicated that the two hotspots (distance 15 kb) mutually inhibit each other to a significant extent for inter- and intragenic recombination. In addition the hotspots showed competition (deficit of double events at both hotspots) which was a minor part of the overall reduction. When histone acetyl transferases Gcn5 and Ada2 were eliminated, recombination at both hotspots was reduced, and mutual inhibition abolished.