In Schizosaccharomyces pombe, cytokinesis initiation and progression is governed by a group of protein kinases called the Septation Initiation Network (SIN). These proteins are recruited and progressively activated on spindle pole bodies (SPB). Interestingly, although in initial mitotic phases some SIN proteins are distributed symmetrically on both SPBs, after sister chromatid separation they become enriched on only one SPB, while fading on the other. This leads to SPB asymmetry at the end of mitosis, which may be preserved after cell division.

It is known that the SIN activity is regulated by nucleotide status of the signaling GTPase, spg1p, whose nucleotide status is controlled by the GAP, cdc16p-byr4p and a putative GEF, sid1p. We are combining automated image analysis and in silico modeling to determine the rules governing the transition of SIN proteins from the symmetric to asymmetric state, which correlates with activation of the SIN. We also plan to extend this analysis to murine cells, to study the consequences of interfering with this control circuitry.

**Conclusions:**
- cdc7 appears after SPB separation on both SPBs. Symmetric localization correlates with short spindle.
- The levels of cdc7 when symmetric on both SPBs are moderate.
- After sister chromatid separation, cdc7 becomes gradually enriched on nSPB and fades from cSPB during spindle elongation.
- sid1 appears on nSPB at the beginning of anaphase and its levels increase gradually with spindle elongation.
- byr4 appears on nSPB and shows similar behavior to that of sid1 on nSPB.
- Establishing rules in wild-type cells will help in screen for proteins that affect SIN status and cell cycle control.

**Future perspectives:**
- Establishment of SIN kinetics in mutant genetic backgrounds.
- Development of software enabling semi-automated analysis of SIN kinetics in collaboration with Prof. M. Unser.
- Development of quantitative model of SIN behavior in collaboration with Dr. I. Xenarios.