8 SUMMARY

The non-essential *Saccharomyces cerevisiae* protein Isw2 is a subunit of the ATP-dependent chromatin remodelling complex Isw2 that regulates the structure of chromatin, and thus plays an important role in regulation of transcription.

Absence of the Isw2p induces the α-mating type-specific aberrant "shmoo-like" morphology and invasive growth. We showed that this phenotype is caused by de-repression of **a**-specific genes in *MAT*α cells and production of inappropriate **a**-factor which in turn activates the pheromone response pathway of *isw2*Δ *MAT*α cells. Our results showed that *isw2*Δ *MAT*α cells can express the *MAT***a**-specific genes at the level which enables them to mate with other *MAT*α cells. Invasion of *isw2*Δ *MAT*α cells depends neither on the cell surface protein Flo11, nor on the invasive growth-specific transcription factor Tec1. On the other hand it requires components of the pheromone response pathway and surface protein Fig2. Together with the fact that the *isw2*Δ *MAT*α cells produce the **a**-factor, the *isw2*Δ-induced invasion resembles the invasive growth commonly occurring in wild-type cells under low concentrations of pheromone. Detailed analysis however uncovered some differences since the *isw2*Δ-invasion depends on the Fus3 kinase (which cannot be substituted by Kss1p) and on the Aga1 cell surface protein which was not found to be important for the pheromone-induced invasive growth.

We identified an interesting phenotype of *isw2*Δ cells – budding within the birth scar, area that is commonly prohibited for budding. Based on our microarray data and double deletion mutant analyses, we identified one gene that is responsible for this phenotype. Deletion of the *DSE1* gene abolished the aberrant budding of the *isw2*Δ cells as well as suppressed the CWI pathway activation described previously for the *isw2*Δ cells (Ruiz, et al., 2003). Consistently, the plasmid-driven overexpression of the *DSE1* gene in wild-type cells induced the budding-within-the-birth-scar phenotype as well as CWI pathway activation. These data prove, that de-regulation of expression of the daughter cell-specific gene *DSE1* is responsible for the CWI pathway activation and aberrant budding occurring in the *isw2*Δ cells.