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The fission yeast *Schizosaccharomyces pombe* has two mating types, called P (plus) and M (minus). A single cell with P or M mating-type newly germinated from a spore forms a haploid population of nearly the equal P/M ratio by mating-type switching (MTS) during repeated cell divisions. MTS follows the so-called 'Miyata's rules' inferred from pedigree analyses, which contain the 'one-in-four rule' and 'recurrent switching rule'. The mating type is determined by genetic information expressed from the *mat1* locus: *mat1-P* in P cells and *mat1-M* in M cells. The *mat1-P/M* gene is replaced efficiently by one of the two silent donor cassettes, *mat2-P* and *mat3-M* by the Rad51–Swi2–Swi5-driven gene conversion. The correct donor choice requires two competing small Swi2-dependent recombination enhancers, *SRE2* and *SRE3*, that function in the context of heterochromatin. Although many studies have provided molecular insights into the MTS mechanism, it has not been completely elucidated yet. Especially, the donor choice mechanism is still largely enigmatic. In this study, first I performed a genetic screening to identify yet-unknown MTS genes and analyzed their functions for donor choice during MTS.

To this end, I prepared a gene deletion library that consisted of homothallic derivatives of the Bioneer ver. 2 library. By multiplex PCR and iodine staining assay, I identified shf1 as a yet non-annotated MTS gene. Sfh1 is one of the subunits of HULC (histone H2B ubiquitin ligase complex).

The structural analysis of the *mat1* locus by Southern blot indicated that *shf1Δ* had no detectable defect in the DNA double-strand break formation required for the initiation of the MTS gene conversion. On the other hand, the donor selectivity assay using h^{09}, in which the *mat*2 and *mat3* cassettes were swapped with each other, showed a defect of *shf1Δ* in the donor selection step of the MTS process. Further mutational analysis with various *SRE* mutants suggested the involvement of Shf1 in the usage of *SRE3* in the M cells.

Since the HULC-mediated H2B ubiquitination function is required for the H3K4 mono-, di- and tri-methyltransferase complex Set1C/COMPASS, and Iwasaki's lab previously isolated mutants of Set1C as MTS mutants, I analyzed also *set1* mutants by the same assays described above, suggesting HULC and Set1C function in the same step in the MTS process.

ChIP-qPCR analysis revealed that in both the *shf1*Δ and *set1*Δ backgrounds, the high, M-specific, Swi6 occupancy at *SRE2* and *SRE3* was decreased. Another ChIP-qPCR analysis suggested that *shf1*Δ and *set1*Δ mutation affected H3K9me2 and -me3 accumulation in the *mat* silenced region.

Taken together, my results suggest that HULC and Set1C inhibit the use of the *SRE3* recombination enhancer in M cells, thus favoring *SRE2* and *mat2-P*.

Based on all of the observations obtained from previous and current studies, I propose a model for the regulation of MTS. In M cells, HULC and Set1C fine-tune the organization of Swi6-containing chromatin. The Swi2–Swi5 complex, a mediator of Rad51-driven strand exchange, associates with both *SRE2* and *SRE3* but its action at *SRE3* is counteracted by the established chromatin structure, resulting in preferential use of the competing *SRE2* enhancer. In P cells, the Swi2–Swi5 complex localizes at *SRE3* specifically and promotes *mat3-M* donor choice, as suggested previously.