Project 7 (P7): Regulation of the dimorphic transition in *Schizosaccharomyces japonicas*
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Project Summary

The ability to alter the growth form in response to environmental conditions is an important virulence associated trait of pathogenic fungi which helps the pathogen to spread in and survive the host's defense system. Such an alteration of the growth form in response to extrinsic signals is not limited to pathogenic fungi but is also found in the model yeasts such as the fission yeast *Schizosaccharomyces pombe*. In response to specific extrinsic cues *S. pombe* cells can leave the vegetative single celled growth mode and form invasive pseudohyphae. Our analysis shows that the *S. pombe* Asp1 protein, which is a member of the conserved Vip1 1/3 inositol polyphosphate kinase family is a key regulator of the morphological switch. An increase in the cellular amounts of Asp1 generated inositol pyrophosphates increases the cellular response thus implying that these molecules might act as second messengers. As the budding yeast Asp1 ortholog is also required for the dimorphic switch in this yeast, we propose that Vip1 family members have a general role in regulating fungal dimorphism.

In this project, we will extend our analysis of Vip1 family function to *Schizosaccharomyces japonicus*. This fungus is non-pathogenic, closely related to *S. pombe*, dimorphic and can change between unicellular and hyphal growth. It is thus a suitable system to study the molecular requirements needed for the transition to and maintenance of the hyphal growth form. The following questions will be answered in this project: Are members of the Vip1 family responsible for the dimorphic switch in *S. japonicus*? If this is the case then how do Vip1 proteins control this switch on the molecular level and what is the impact of the actin and microtubule cytoskeletons? Are members of the Vip1 family universal regulators of cellular morphogenesis? Do they regulate the dimorphic switch in pathogenic fungi such as *C. albicans* and *U. maydis*? We expect that our analysis of Vip1 function in various fungi will help to understand if dimorphic events are regulated by a common Vip1 pathway.