

BIG seminar

Biology and integrative genetics

Monday, January 18, 17h

Auditorium Bâtiment Biophore
Unil-Sorge at Dorigny

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“Next-generation genetics in plants: Evolutionary tradeoffs, immunity and speciation”

We are addressing three core questions:

- How, and how frequently, do new genetic variants arise?
- Why do some variants increase in frequency?
- Why are some combinations of new variants incompatible?

These correspond to the fundamental evolutionary processes of mutation, selection and speciation, which we are studying using both bottom-up (i.e., forward genetic) and top-down (i.e., whole-genome) approaches. I will begin by showcasing examples that demonstrate the power of second-generation sequencing, both in support of forward genetics (Schneeberger et al., *Nature Methods* 2009), and in determining the rate and spectrum of mutations in the plant *Arabidopsis thaliana* (Ossowski et al., *Science* 2010). Based on our experience with short-read sequencing (Ossowski et al., *Genome Research* 2008), we have been advocating a 1001 Genomes project for *A. thaliana* (Nordborg & Weigel, *Nature* 2009; <http://1001genomes.org>), and we have already sequenced 84 wild strains from this species. I might briefly touch on some of the results from these efforts as well. Next, I will discuss a fitness trade-off we recently discovered. The inconstancy of the environment places organisms under competing evolutionary pressures, particularly sessile organisms like plants. Allelic variants beneficial in one setting might be detrimental under different circumstances. Plants vary greatly in their ability to resist microbial or animal attack, and this is thought to reflect fitness costs in the absence of pathogens or predators. We have found that allelic diversity at a single locus, *ACCELERATED CELL DEATH 6* (*ACD6*), underpins dramatic variation in both vegetative growth and resistance to microbial infection and herbivory in *A. thaliana*. Another case of a potential evolutionary trade-off is provided by hybrid necrosis, an autoimmune syndrome that has been observed in many intra- and interspecific plant crosses. Several independent *A. thaliana* cases result from epistatic interactions that trigger autoimmune-like responses, and that behave as expected for Dobzhansky-Muller incompatibilities (Bomblies & Weigel, *Nature Reviews Genetics* 2007; Bomblies et al., *PLoS Biology* 2007). Molecular analysis has revealed two recurrent themes: the causal genes often occur in polymorphic clusters and most encode disease resistance genes, which explains the autoimmune phenotype. Our findings implicate the extreme allelic diversity of disease resistance genes (Clark et al., *Science* 2007), presumably due to pathogen pressures, as potential causes for the evolution of gene-flow barriers in plants.

