

genetic change of blast fungus of grass around the rice field when they passed through different host genome. Assessment of genetic change during host alteration was investigated through the genetic analysis of the basal samples of isolated *Pyricularia* from *Digitaria ciliaris* after infection this isolate into rice and *Panicum repens* using SCAR markers (Cut1, Erg2, PWL2), rep-Pot2, Amplified fragment length polymorphism (AFLP) and pathotype. The result of this study showed that cross infection of *Pyricularia* d4 from *D. ciliaris* grass into rice induced genetic variation in their Cut1 and PWL2 markers, AFLP and rep-Pot2 patterns, as well as pathotype. The infection into susceptible rice plant could induced genetic change more higher than into resistant plant. However, the infection into different genus of grass (*P. repens*) induced genetic change only in AFLP and rep-Pot2 patterns, but not in SCAR markers. Result of this study indicated that the cross infection in different host genome might induce microevolution of *Pyricularia* d4.

PS20-617

Evolutional origin of the conditionally dispensable chromosomes controlling pathogenicity of *Alternaria alternata* pathogens

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The filamentous fungus *Alternaria alternata* includes seven pathogenic variants (pathotypes), which produce host-specific toxins (HST) and cause necrotic diseases in different plants. The HST biosynthetic genes have been isolated from six pathotypes (Japanese pear, strawberry, tangerine, apple, tomato and rough lemon) of *A. alternata*. The genes for biosynthesis of each toxin are located at the same locus in the genome, defining a gene cluster. The HST biosynthetic gene clusters of the six pathotypes were found to reside on single small chromosomes of <2.0 Mb in most strains tested. Loss of the small chromosomes was observed in the strawberry, apple and tomato pathotypes, and the small chromosomes appeared to be conditionally dispensable (CD) chromosomes. We determined the structures of the CD chromosomes of the strawberry, apple and tomato pathotypes and identified putative toxin biosynthetic gene cluster regions on the chromosomes. Pairwise comparison of the entire regions of CD chromosomes from these pathotypes identified large syntenic regions among the three CD chromosomes. The regions including HST gene clusters are unique to the respective pathotypes, but the remaining regions of CD chromosomes from the three pathotypes are conserved. The co-linear order of genes has been maintained within the CD chromosomes of the three pathotypes. These results suggest that the CD chromosomes have a common origin, and that the syntenic regions are the core of the original, dispensable chromosome.

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Genome evolution of fungal pathogens from *Magnaporthe oryzaelgrisea* clade

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Magnaporthe oryzae is a fungal species complex gathering pathogens of different Poaceae that causes the main fungal disease of rice and severe epidemics on wheat in South America. This project aims at characterizing genomic determinants and evolutionary events involved in the adaptation of fungi to different host plants. Eight strains from *M. oryzae* species complex pathogenic on either rice, wheat, *Setaria* or *Eleusine* and one strain of the related species *M. grisea* pathogenic on *Digitaria*, have been sequenced using NGS. De novo annotation was carried out with Eugene for gene and with REPET for transposons. Most frequent families are LTR retro-transposons, but some DNA transposons were found. Repeats cover about 10-12% of these genomes. Variable genome sizes (36-42 Mb) and gene contents (12 300-20 500 genes) were estimated for these genomes, even though 4 genomes were more fragmented (poor scaffolding, short and truncated CDS). OrthoMCL analysis including *M. oryzae* 70-15 reference genome, identified 20 443 clusters, including 8 154 single copy shared by-all families (core genome) and variable number of species-specific gene families (305-1550). Gene families expected to be involved in pathogenicity including genes encoding enzymes involved in the biosynthesis of secondary metabolites, enzymes involved in plant cell wall degradation and small secreted peptides are currently analyzed. 12-14% of the predicted CDS encode putative secreted proteins with a median length of 260 aa. A dedicated database was developed to facilitate evolutionary analyses and integration of RNAseq data from in planta infection kinetics. Additional comparative analyses will be presented.

PS21-619

Structural insights into TIR domain and effector function in effector-triggered immunity in flax and Arabidopsis

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Effector-triggered plant immunity is initiated through the recognition of a pathogen effector protein by a plant resistance (R) protein, leading to the activation of plant defenses and a localized cell death response. The effectors usually have roles in virulence and are structurally diverse, while R proteins generally fall into a few conserved families. We have used the fungal pathogen flax rust interaction with flax as a model system to characterize this process. The flax R proteins consist of a core nucleotide-binding domain, an N-terminal Toll-interleukin 1-receptor (TIR) domain, and a C-terminal leucine-rich repeat (LRR) domain. We have shown the direct interaction of the effector proteins AvrL567 and AvrM with R proteins L6 and M, respectively, and also determined the crystal structures of AvrL567 and AvrM. Recently, we determined the crystal structure of a TIR domain from L6 at 2.3 Å resolution. The structure reveals important differences from the structures of mammalian TIR domains, and highlights three separate functionally important protein surfaces, involved in oligomerization, interaction with a downstream signaling partner, and regulatory intramolecular interactions, respectively. We have also complemented this work with a study of the TIR domains of Arabidopsis proteins RPS4 and RRS1, which work in concert to confer resistance to several pathogens. Our results bring us a step closer to understanding the molecular basis for the disease resistance process.

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Structure analysis of Tomato spotted wilt virus nucleocapsid proteins

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