What drives speciation? Examination into the evolutionary events of more than 100 Aspergillus species

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Publication date:
2017

Document Version
Publisher's PDF, also known as Version of record

Saturday, March 18 2:00 PM – 5:00 PM
Kiln
Chromosome Dynamics

What drives speciation? Examination into the evolutionary events of more than 100 *Aspergillus* species. *J.L. Nybo*[1], T.C. Vesth[1], S. Theobald[1], I. Kjaerboelling[1], J.C. Frisvad[1], T.O. Larsen[1], R. Riley[2], A. Salamov[2], I.V. Grigoriev[2], S.E. Baker[3], M.R. Andersen[1] 1) Biotechnology and Biomedicine, Technical University of Denmark, Kongens Lyngby, DK; 2) Joint Genome Institute, Walnut Creek, CA, USA; 3) Joint Bioenergy Institute, Berkeley, CA, USA.

The study of speciation - how new species arise, diverge and remain separate, has a central role in evolutionary biology. Partly because it embraces so many disciplines, including population genetics, behavioral sciences, comparative genomics, evolutionary biology, biodiversity, biogeography and ecology. It also remains one of the most fascinating questions in evolution.

Speciation is nearly impossible to study and in most cases, we know very little about the genetic basis of species formation. But in this project we aim to identify evolutionary events that can drive speciation, such as gene duplications, creations and losses, and horizontal gene transfers between closely or distantly related species within the genus of the filamentous fungi Aspergillus. This diverse genus holds species relevant to both plant and human pathology, food biotechnology, enzyme and bulk chemical production, model organisms, and it even contains some extremophiles.

To identify these events, we have developed a homologous protein prediction software that has been used to generate a high-resolution pan-genomic map. From where, we have identified genes specific to species, clades and core that allows for guilt-by-association-based mapping of genotype-to-phenotype.

Our results illustrate a highly diverse genus where 500-2000 genes are unique to each species. These genes are predominantly within regulation or compound biosynthesis, supporting the notion of natural selection. A conservative estimate of the number of protein families shared by all Aspergillus species is surprisingly low, only about 2600 core families, suggesting high environmental adaptation within this genus.

Transitions between tetrapolar and bipolar fungal mating type driven by chromosomal translocations involving intercentromeric recombination. *Sheng Sun*[1], Vikas Yadav[2], R. Blake Billmyre[1], Christina A. Cuomo[3], Minou Nowrousian[4], Jean-Luc Souciet[5], Teun Boekhout[6], Betina Porcel[7], Patrick Wincker[7], Joshua A. Granek[1], Liuyang Wang[1], Kaustuv Sanyal[2], Joseph Heitman[1] 1) Molecular Genetics & Microbiology, Duke University Medical Center, Durham, NC; 2) Molecular Biology and Genetics Unit, Jawaharlal Nehru Centre for Advanced Scientific Research, Bangalore, India; 3) Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA; 4) Lehrstuhl für Allgemeine und Molekulare Botanik, Ruhr-Universität Bochum, Bochum, Germany; 5) Université de Strasbourg, CNRS UMR7156, Strasbourg, 67000, France; 6) CBS-KNAW Fungal Biodiversity Centre (CBS-KNAW), Utrecht, The Netherlands; Institute of Biodiversity and Ecosystem Dynamics (IBED), University of Amsterdam, Amsterdam, The Netherlands; 7) Commissariat à l'Energie Atomique (CEA), Institut de Génomique (IG), Genoscope, Evry, France, Université d'Evry, UMR 8030, Evry, France, Centre National de Recherche Scientifique (CNRS), UMR 8030, Evry, France.

Species within the human pathogenic *Cryptococcus* species complex are major threats to public health, causing more than one million infections globally each year. *Cryptococcus amylolentus* is the most closely known related species of the pathogenic *Cryptococcus* species complex, and it is non-pathogenic. Additionally, while pathogenic *Cryptococcus* species have bipolar mating systems with a single large *MAT* locus that represents a derived state in Basidiomycetes, *C. amylolentus* has a tetrapolar mating system with two *MAT* loci (*P/R* and *HD*) located on different chromosomes. Thus, studying *C. amylolentus* could shed light on the origin and evolution of pathogenesis, as well as the transition from tetrapolar to bipolar mating systems in the pathogenic *Cryptococcus* species. In this study, we sequenced, assembled, and annotated the genomes of two *C. amylolentus* isolates, CBS6039 and CBS6273, which are interfertile. Genome comparison between the two *C. amylolentus* isolates identified the boundaries and the complete gene contents of the *P/R* and *HD* loci. Also, bioinformatics and ChIP-seq analyses showed that *C. amylolentus* has regional centromeres that are enriched with species-specific transposable and repetitive elements, similar to the centromeric structures in the pathogenic *Cryptococcus* species. Additionally, we found that while neither of the *P/R* and *HD* loci in *C. amylolentus* is physically linked to its centromere, both *MAT* loci showed centromere linkage in meiosis, suggesting the presence of recombination repressors and/or epistatic gene interactions in the inter *MAT*-CEN regions. Furthermore, genomic comparison between *C. amylolentus* and pathogenic *Cryptococcus* species provided evidence that chromosomal rearrangements mediated by intercentromeric recombination have occurred after the two lineages split from their common ancestor. We propose a model in which the evolution of the bipolar mating system was initiated by an ectopic recombination event mediated by repetitive elements located within the centromeric regions and shared between chromosomes. This translocation brought the *P/R* and *HD* loci onto the same chromosome, and was followed by chromosomal rearrangements that resulted in the two *MAT* loci becoming physically linked and eventually fused to form the single contiguous *MAT* locus that is now extant in the pathogenic *Cryptococcus* species.

Genome plasticity impacts adaptive genome evolution in the vascular wilt pathogen *Verticillium*. *M.F. Seidl*, L. Faino, D.E. Cook, M. Kramer, X. Shi-Kunne, G.C.M. van den Berg, B.P.H.J. Thomma Laboratory of Phytopathology, Wageningen University & Research, Wageningen, NL.

Genome plasticity enables organisms to adapt to environmental changes and to occupy novel niches. This is established by mechanisms ranging from single-nucleotide polymorphisms to large-scale chromosomal variations, all of which contribute to differences in chromosomal size, organization and gene content. While these mechanisms operate in all organisms, they are particularly relevant for plant pathogens that engage in a co-evolutionary arms race with their hosts. Plant pathogens secrete so-called effectors that contribute to host colonization and counteract host immunity. Effector genes often cluster in highly plastic, transposon-rich genomic regions. However, mechanistic understanding of the evolution of these plastic genomic regions remains scarce. We study these molecular mechanisms in the fungal genus