

Current Trends in Fungal Genomics and Biology

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The deliberate decision in the early 1940s to use *Neurospora crassa* in the Nobel Prize winning work that demonstrated an association between genes and enzymes initiated an avalanche of research now known as Molecular Genetics. Among the reasons Beadle and Tatum chose a fungus for that seminal work included the facts that while *Neurospora* is a eukaryote, it can be rapidly propagated from asexual conidia or mycelial fragments, with each culture providing millions of genetically identical haploid nuclei. Further, strains of opposite mating type can readily be crossed in order to analyze the inheritance pattern of mutant traits. When added to the fact that many fungi are pathogenic to plants and animals and contain much less DNA per genome than higher eukaryotes, it is somewhat surprising that the only fungal genome sequenced before the human genome was released in 2001 was that from a strain of *Saccharomyces cerevisiae* [1]. However, as rapid developments in technology have greatly reduced the expense of sequencing, fungal genomics has literally exploded. As of this writing, 353 fungal and 20 oomycete species are listed in the 'genome completed' section of The National Center for Biologic Information (NCBI). A '1,000' Fungal Genomes Project initiated at the end of 2010 promises to add many more in short order (see <http://genome.jgi-psf.org/programs/fungi/1000fungalgenomes.jsf>).

In quite a few cases, multiple species in the same or closely related genera are now available. Not surprisingly, groups that include species are pathogenic or of economic value to humans are most often sequenced: sequences are now available for the genomes of 10 species of *Saccharomyces*, 9 of *Candida* and 6 each of *Schizosaccharomyces* and *Penicillium*. Among those fungi that are primarily associated with plant diseases, there are sequences for 6 species of *Phytophthora*, 5 of *Fusarium* and 5 of *Cochliobolus*, many of which are pathogenic to different host species. Once the DNA sequence of the first species in a genus has been deciphered, the syntenic relationships of genes and chromosomes usually makes genome assembly much simpler for those that follow. Because each of the previously mentioned groups contains both pathogens and non-pathogens, most comparisons to date have focused on identification of genes unique to the pathogens [2-11]. Typical results reveal logical outcomes, such as the presence of multiple genes encoding secreted proteins or expanded gene families that broaden the amount or array of degradative enzymes produced. In other cases, secondary metabolites or regulatory elements such as G-proteins or specific kinases differ significantly. A common feature is to find that the genomic differences between pathogens and non-pathogens can be traced to 'islands' of transposon-containing sequences in the pathogen DNA, suggesting gene duplication and following changes have contributed to the evolution of virulence [7,12]. Even though the genomic differences make sense in terms of biological effects, it also seems that most of the differences documented so far are unique to each comparison. Whether that proves to be the case as more and more genomic comparisons are made, or if a set of fungal genes can be identified that are common to pathogenic species, it can be anticipated that the knowledge gained will eventually aid in developing disease resistant hosts [13].

As more genomes become available, additional factors involved in differences in pathogens also become targets for comparison. In

an attempt to identify genes that determine host specificity, Richard Michelmore is taking advantage of his position as the Director of the UC Davis sequencing facility to compare the genomes of downy mildews that attack important crops, including both monocots and dicots. Another area of promise involves identification of the fungal 'elicitors' and 'effectors' that trigger or prevent host defense responses. In comparison to bacteria, relatively little is known concerning identify of molecules that trigger recognition by the host. Many plant pathogenic fungi show 'gene for gene' interactions (at the protein level) with the host: interaction of a fungal protein and a host receptor coded by a resistance gene triggers a cascade of events that can prevent reproduction of the pathogen (resistance). Techniques such as RNA-Seq, microarrays and two-hybrid assays with data collected at various times after inoculation from both incompatible (resistance) and compatible (disease) interactions should eventually pinpoint the genes involved for both the pathogen and the host. Natural mutants or knock-outs of fungal genes identified as virulence factors will elucidate the basis for interactions with specific R (resistance) genes in the host. Ultimately, sequence differences will reveal the basis for unique 'pathotypes' or 'races', i.e., strains that differ in the ability to cause disease when a particular resistance gene is present. Difference between pathogens with a broad host range such as *Macrophomina phaseolina* and those with strict host specificity should also be revealed via genome comparisons. A recent multi-authored paper [9] was able to associate sequence differences in two closely related pathogens with basic differences in symptoms. *Sporisorium reilianum* and *Ustilago maydis*, are both smut fungi that can cause disease on maize. While *S. reilianum* becomes systemic on infection, *U. maydis* causes local tumors at the site of infection. Identification and comparison of regions in the two genomes that have relatively low sequence homology were informative. For the most part, the variable sequences still encode proteins that are secreted; the fact that they differ significantly in interactions with the host is assumed to account for their evolution into separate species.

Practical applications are driving further research in fungal genomics. One benefit readily gleaned from genomic sequencing is the presence of modules that encode non-ribosomal peptide synthetases; the peptides made often have antimicrobial activity that makes them of potential value as pharmacological agents. Similarly, the presence of genes that encode enzymes in the pathways for synthesis of mycotoxins can be identified. Comparisons between strains that produce the toxins and those that do not in specific environments are expected to provide clues for control, thus providing a safer food supply. Likewise, the

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presence or absence of genes required for RNAi can be determined, providing potentially useful information on prospects for the use of novel control methods in genetically engineered hosts. Finally, since wood rotting fungi are a rare source of enzymes with the ability to degrade lignin, considerable interest has developed for identifying the critical genes and their gene products for use in enhanced production of biofuels.

On the basic science side, newly developed non-toxic fluorescent probes being used to tag and follow gene expression in real time are providing insights into the mechanisms of growth and development that have implications extending beyond simple fungi.

As I often tell my students, it is really an exciting time to be working in genetics. Fortunately, it has been true every year of my 40-plus years of working with fungi.

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