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Study takes aim at opportunistic fungal pathogens

Research provides genome sequences and critical analyses of key Candida species

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In what represents one of the largest comparative genomics studies to date, scientists have cracked the genetic code of several fungal species that cause bloodstream infections in patients with suppressed immune systems.

The international team, which includes researchers from the Broad Institute of Harvard and MIT, decoded and analyzed the genomes of six species of Candida, a group of fungi related to the harmless baker's yeast. The findings, published online last month in the journal Nature, significantly extend the arsenal of genomic information on these organisms and offer some initial clues about what makes some fungi pathogenic and others not.

"Most of what we know about Candida comes from the study of a single species, Candida albicans," said Christina Cuomo, co-senior author of the study and a research scientist at the Broad Institute. "But there are at least six other species that together account for nearly half of all Candida infections. Our work is a key step toward deepening our knowledge of these medically important fungi."

Candida are the most common cause of invasive fungal infections, with an average mortality rate of about 40 percent. At present, the treatment of fungal infections is difficult. Several antifungal drugs can be very effective in treating such infections, but not always. Moreover, because fungi are eukaryotes and therefore have many genes that are similar to those in humans, designing drugs to specifically target fungi is more difficult than for bacteria.

"In order to understand how to treat the infection we first have to understand how the fungi interact with the host," said Geraldine Butler of University College Dublin, one of the lead scientists involved in the research. "Our findings will help scientists to develop potential therapies for the acquired infection."

The researchers chose six Candida species for whole-genome sequencing that include both strong pathogens and less virulent species, and compared these strains to 11 previously sequenced fungal genomes. By recreating the gene trees for thousands of gene families and studying the patterns of gene gain and loss, the scientists found that the disease-causing species have many more genes associated with the cell wall, and genes that enable them to successfully stick to the host. These fungi also secrete more enzymes that can help them to invade host tissue.

"We found that some gene families showed dozens of gene duplication

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events in the pathogenic species, but none in the non-pathogens," said co-senior author Manolis Kellis, the Karl Van Tassel Career Development Associate Professor of Computer Science and associate member of the Broad Institute. "Some of these families are still uncharacterized, but they contain cell-surface adhesion domains typically involved in pathogenicity, providing some clues to their function."

The authors used the new genomes to discover new genes in Candida albicans based on evolutionary signatures of protein-coding selection, revealing many novel genes that are unique to the Candida species, and to study mating and meiosis pathways, revealing extensive diversity that challenges previous models developed in yeast.

The new work is part of the Fungal Genome Initiative at the Broad Institute and was funded by the National Human Genome Research Institute and the National Institute of Allergy and Infectious Diseases among other organizations. In addition to Cuomo and Kellis, researchers from MIT and the Broad Institute who contributed to this research include Matthew Rasmussen, Michael Lin, Bruce Birren, Manfred Grabherr, Chinnappa Kodira, Sharadha Sakthikumar and Qiandong Zeng, and members of the Broad Institute's Genome Sequencing Platform.