QUANTITATIVE & SYSTEMS BIOLOGY COLLOQUIUM:

Date: 1/29/2026
Time: 10:30 AM – 11:45 AM
Location: COB1 282

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Deepika Gunasekaran, Postdoctoral Scholar

Genomic Super-enhancer-like Features Regulate Cell Fate in Candida albicans

Abstract:

Candida albicans, an opportunistic pathogen, is one of the few fungal species that undergoes reversible, heritable switching between white and opaque morphologies which is hypothesized to contribute to its pathogenesis. In higher-eukaryotes, cell fate transitions are regulated by non-coding elements called "super-enhancers", characterized by the presence of high densities of transcription factors, histone acetylation marks and occupancy by core transcriptional machinery. In our study, we examined whether these features are associated with cell fate in C. albicans using an integrative approach, combining genome-wide binding of core white-opaque regulators and transcriptional mediators, acetylation of lysine 27 on histone H3 (H3K27ac), chromatin accessibility and gene expression. We identified super-enhancer-like (SE) elements using Mediator complex occupancy and show that they are associated with increased RNA polymerase II occupancy, H3K27ac, and neighboring gene expression. We found that six of the eight core white-opaque regulators are regulated by SE elements and 80% SE elements are occupied by at least one of these regulators. SE elements are more accessible with increased binding of core white-opaque regulators. Even though we identified SE elements in both cell types, they regulate cell-type specific functions, i.e. phenotypic switching in opaque, and housekeeping functions in white cells. Additionally, 70% genes regulated by SE elements are highly conserved. We show, for the first time, the role of SE elements in regulating cell fate in C. albicans despite two-thirds of its genome being protein-coding. Our findings indicate that white-opaque regulators drive cell fate switching in a concerted manner through co-localization in regulatory regions.

About The Speaker:

Deepika Gunasekaran is a postdoctoral scholar at UC Merced in Dr. Clarissa Nobile's lab. She is a computational biologist whose research focuses on developing methods to study the regulation and evolution of gene expression. She earned her PhD at UC Merced, where she was co-advised by Dr. Clarissa Nobile and Dr. David Ardell, and studied the evolution of transcriptional networks controlling biofilm formation in fungal pathogens. Her current research combines computational and machine-learning approaches to investigate transcriptional regulation, pathogenesis, and drug tolerance in fungi.

Namkha Nguyen, Graduate Student

Orchestration of Transcription Factors and Epigenetic Modifiers in Candida Albicans Cell Plasticity

Abstract:

Understanding how cells differentiate and heritably maintain their identity is a fundamental challenge in cellular biology. Candida albicans is a polymorphic diploid fungus that can epigenetically switch between two distinct cell types: "white" and "opaque." These phenotypes are maintained over hundreds of generations, yet switching between them can occur stochastically. The white-opaque switch has emerged as a powerful and relatively simple model for studying cell plasticity, as its high-dimensional, interwoven transcriptional circuitry bears a striking resemblance to regulatory networks governing complex developmental processes in higher eukaryotes. Notably, equivalent programs are absent in other model yeasts such as Saccharomyces cerevisiae. Sequence-specific DNA-binding proteins, or transcription factors, have been the primary focus in defining the white-opaque transcriptional network, which left regulation of chromatin accessibility, also a critical determinant of gene expression, relatively understudied. My work seeks to uncover the roles of chromatin modifiers in the white-opaque switch and how they coordinate with transcription factors to expand our understanding of cell-fate decision making.

About The Speaker:

I began my research career during my freshman year as an undergraduate at UC Merced with Dr. Aaron Hernday, where I developed a rapid, efficient, and recyclable CRISPR-Cas9 genome-editing system for Candida albicans. This approach enabled simultaneous deletion of both alleles of up to three genes, addback of deleted genes at their native loci, and iterative editing within the same strain. I subsequently applied this system to optimize allele-specific editing to investigate potential echinocandin drug targets. Building on this interest in CRISPR-based tool development, I continued my graduate training with Dr. Hernday, developing a locus-specific pulldown strategy to identify chromatin-associated factors in C. albicans as well as a genome-editing platform for the endemic fungal pathogen Coccidioides spp. In the latter part of my graduate work, I have used chromatin profiling techniques to investigate the mechanisms underlying the white-opaque switch. Outside the lab, I enjoy outdoor recreation, chess, video games, and making music.

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