Temple University
Department of Biology

-Final Doctoral Thesis Defense-

TITLE

"SELECTIVE FORCES SHAPING DUPLICATE GENE EVOLUTION: INSIGHTS FROM STOCHASTIC MODELING AND PATTERNS OF RETENTION"

Amanda Erin Wilson

TIME AND PLACE

Tuesday, April 2nd, 2024
9:00 AM
Bio-life Building, room 234
And Via Zoom link - https://temple.zoom.us/j/93781104584

Light refreshments starting at 8:30 am
Any questions, please contact the Biology Department @ 215-204-8854

Dissertation Committee
Dr. David A. Liberles, Advisory Committee Chair, Temple University
Dr. Sergei Pond, Examining Committee Chair, Temple University
Dr. Rachel Spigler, Member, Temple University
Dr. Anne-Ruxandra, External Member, University of Pittsburgh

Abstract: The variation of genome content and structure across the tree of life is astounding and can provide clues to understand the process of evolution. Overall, this helps us understand the history of life and how organisms have fundamentally changed and adapted to their environments. Gene duplication is an important mechanism for molecular evolution because it provides opportunity for functional novelty and molecular innovation. Gene duplication creates new functional gene copies with different selective pressures that allow them to take on new or specialized functions. Throughout this work, I explored the interplay between genetic changes, molecular phenotype and selection of duplicate gene copies. I particularly focused on the genetic opportunity, consequences, and selective pressures of the mechanisms of short-term and long-term duplicate copy retention. I modeled the stochastic processes of mutation and selection and their effect on duplicate gene copy retention. Specifically, I modeled the interplay between subfunctionalization and dosage balance and found that selection may cause genes that are sensitive to dosage balance effects to experience delayed subfunctionalization, but ultimately lead to higher levels of subfunctionalization. These findings suggest that subfunctionalization may not occur as a purely neutral process. Next, I used survival analysis methods to model patterns of duplicate gene retention in genomes experiencing consecutive whole genome duplication events. I modeled three hypotheses to explain patterns of duplicate gene retention including the Independence Hypothesis, the Gene Duplicability Hypothesis, and a novel Mutational Opportunity Hypothesis. The results show that under the Gene Duplicability and Mutational Opportunity hypotheses, the expected patterns of duplicate gene retention after consecutive whole genome duplication events is greatly affected by the ages of the whole genome duplication events and the functional properties of the genomic content that influence opportunity and selection. Additionally, I used my models to investigate the patterns of retention in real world phylogenetic datasets. These preliminary results suggest that gene duplicates' retention after whole genome duplication events may be influenced by their sensitivity to dosage imbalance and their functional properties. Key findings underscore the multifaceted nature of duplicate gene retention, influenced by a myriad of factors including genetic opportunity, selective pressures, and evolutionary context. By dissecting the underlying mechanisms driving duplicate gene retention, this dissertation advances our understanding of the evolutionary dynamics shaping genome evolution and functional diversity across diverse biological systems.